JUN 16 1950

MEDICAL LIBRARY

Vol. 3 No. 2 APRIL, 1949

BRITISH JOURNAL OF SOCIAL MEDICINE

EDITORS

F. A. E. CREW and LANCELOT HOGBEN

WITH THE ASSISTANCE OF

ENID CHARLES
G. P. CROWDEN
T. FERGUSON RODGER
J. A. FRASER ROBERTS
LESLIE J. HARRIS
DONALD HUNTER
J. G. KYD
J. R. MARRACK
T. McKEOWN
J. A. RYLE
P. SARGANT FLORENCE
R. S. F. SCHILLING
S. ZUCKERMAN

W. G. CLARK
C. FRASER BROCKINGTON
Representing the Society of Medical Officers of Health

and the EDITOR of the *British Medical Journal*

CONTENTS

	PAGE
Milk-Borne Infections in Great Britain. SIR WILLIAM SAVAGE	45
A Study on Birthweight and Intelligence. CÉCILE ASHER and J. A. FRASER ROBERTS	56
Control Charts for the Standard Mortality Ratio. G. HERDAN	69
Social Medicine. R. C. BROWNE, I. F. BECK and R. I. MCCALLUM	77
Mortality amongst Babies from Injury at Birth. H. T. RUSSELL and IAN SUTHERLAND ..	85
Abstracts	95

LONDON

BRITISH MEDICAL ASSOCIATION

TAVISTOCK SQUARE, W.C.1

YEARLY SUBSCRIPTION (4 NUMBERS) 25s. U.S.A. $6.00 SINGLE NUMBER 7s. 6d.

NOTICE TO CONTRIBUTORS

THE BRITISH JOURNAL OF SOCIAL MEDICINE is intended for the publication of original work in the field, although not necessarily by authors of British nationality.

As understood by those who are responsible for this Journal, social medicine is that branch of science which is concerned with : (*a*) biological needs, interactions, disabilities, and potentialities of human beings living in social aggregates ; (*b*) numerical, structural, and functional changes of human populations in their biological and medical aspects. To a large extent its methods must necessarily be statistical, involving the use of numerical data obtained either from official sources or from special field investigations, and interpreted in the light of established findings of the laboratory and of the clinic. Social medicine takes within its province the study of all environmental agencies, living and non-living, relevant to health and efficiency, also fertility and population genetics, norms and ranges of variation with respect to individual differences and, finally, investigations directed to the assessment of a regimen of positive health. Such being the definition and scope of social medicine as understood by the editors, it follows that this Journal is not meant to provide a platform for those who wish to present their views concerning the place of social medicine in the organizational set-up of medicine as a whole.

The scope of the Journal and, therefore, the content of social medicine will be illustrated by the nature and diversity of the papers that go to the make-up of the first few numbers. These will also show that the territory claimed by social medicine is not adequately covered by journals that serve the interests of the specialists in such allied subjects as public health and preventive medicine, biometrics, eugenics, and the social sciences.

So far as is possible in these difficult days, this Journal will be published quarterly.

All papers submitted for publication and all other editorial matters should be addressed to one of the editors :

F. A. E. Crew, F.R.S., Usher Institute, Warrender Park Road, Edinburgh.

Lancelot Hogben, F.R.S., Queen Elizabeth Hospital, Birmingham 15.

Alternatively papers may be sent in the first instance to any of those whose names appear in the group on the front cover or to any members of the General Advisory Board.

Papers are accepted on the understanding that they are subject to editorial revision, and that they are contributed to this Journal only.

Adequate reference should be made to previous work on the subject of the paper. A summary of observations and conclusions must be given.

Papers sent for publication must be typewritten on one side of the paper only, with double spacing and with a margin of at least 1½ inches. Illustrations should be used sparingly. Photographs should be printed on glossy paper and should be larger than the size desired for reproduction. Drawings and diagrams should be done in black ink on tracing linen, Bristol board, or stout, white paper. Any lettering on these drawings should be lightly inserted in pencil. The Editors express the hope that contributors will study the conventions of shading and design of charts employed in this and subsequent issues of the Journal with a view to standardizing visual symbols for ready recognition.

References should be arranged according to the Harvard system. If the reference is to a book, the place of publication, the number of the edition and the page number should be given. In the text the year of publication must follow the author's name, more than one paper in any one year being indicated by a small letter (*a*, *b*, *c*) after the date. No numbering of references is necessary. At the end of the paper references should be arranged in the alphabetical order of the authors' names. The reference details should be given as follows: author's name, initials, year of publication (in parentheses), title of periodical (in italics, abbreviated according to the *World List of Scientific Periodicals*), volume number (bold type Arabic numerals), and first page number (ordinary type Arabic numerals), thus :

Buzzard, Farquhar (1943). *Practitioner*, **151**, 129.

Grotjahn, A. (1923). *Soziale Pathologie*, 3rd edit., Springer, Berlin.

Contributors will receive one proof, but it is assumed that all but verbal corrections will have been made in the original manuscript; an allowance at the rate of ten shillings per sheet of sixteen pages is made for alterations in the proof (printer's errors excepted), and contributors will be responsible for any excess.

Fifty reprints of papers will, if desired, be provided free. A limited number of additional reprints at cost price can be supplied if application is made when returning proofs. An estimate of the cost will be given on application to the Publishing Manager, British Medical Association.

Papers which have appeared in THE BRITISH JOURNAL OF SOCIAL MEDICINE remain the property of the Journal, and permission to republish them must be obtained from the editors.

Applications for advertisement space should be addressed to the Advertisement Manager, British Medical Association, Tavistock Square, London, W.C.1.

NOTICE TO SUBSCRIBERS

Subscriptions are payable to the British Medical Association. Address: British Medical Association House, Tavistock Square, London, W.C.1. The subscription is 25s. a year to non-members of the B.M.A. and 20s. a year to members of the B.M.A.

Brit. J. soc. Med. (1949), **3**, 45-55.

MILK-BORNE INFECTIONS IN GREAT BRITAIN

BY

SIR WILLIAM SAVAGE

As long ago as 1857 Dr. Taylor of Penrith showed that an outbreak of typhoid fever was spread by milk. Since that date the literature has been strewn with reports on milk-spread outbreaks of infectious disease, but even so numerous outbreaks remain unrecorded.

The importance of milk as a vehicle for the transmission of disease is due to three factors. The cow may suffer from infections which are common also to man; milk is a suitable medium for the multiplication of most pathogenic organisms and ingestion is an important factor in infection; milk is much handled between cow and consumer, so facilitating its infection.

There are three sources of human milk-spread infections. (1) Bovine infection of the cow may cause tuberculosis, brucellosis, some salmonella infections, and, very rarely, foot and mouth disease and anthrax. (2) Through the cow, but in the animal due to human bacterial strains implanted from human sources, some scarlet fever and other streptococcal infections and certain staphylococcal infections are so disseminated. (3) Specific contamination of the milk after it leaves the cow may be the cause of the enteric infections, i.e. typhoid fever, paratyphoid fever and dysentery, diphtheria, some scarlet fever and other streptococcal infections, most staphylococcal infections, and some salmonella outbreaks.

Although it is possible to estimate with considerable accuracy the extent to which tuberculosis in man is of bovine origin and to make an estimate of human brucellosis, we are without reliable statistics of the extent of other infections.

The Ministry of Health list of milk-borne outbreaks of infection reported in Great Britain between 1900 and 1933 and tabulated by the Committee on Cattle Diseases includes 97 outbreaks, analysed as enteric fever 30, paratyphoid fever 7, typhoid and dysentery 4, diphtheria 14, scarlet fever 28, sore throat 5, gastro-enteritis 3, salmonella 3, sickness and diarrhoea 3. G. S. Wilson in *The Pasteurization of Milk* mentions 69 outbreaks in the years 1912-37 comprising scarlet fever and septic sore throat 40, diphtheria 20, the three enteric diseases 39, gastro-enteritis 14; total 113. Both authorities stress the marked incompleteness of the records. The comparative prevalence today of these infections can be judged from the following list of 25 outbreaks over the last seven years collected by the author without any detailed study of the literature. They are (excluding tuberculosis) undulant fever 2, scarlet fever 2, diphtheria 1, typhoid fever 2, paratyphoid fever 4, dysentery 11 (Sonne type 9, other types 2), salmonella Dublin 2, staphylococcus food poisoning 1. Many outbreaks are never recorded in the literature or only obtain brief mention in not readily accessible annual reports of medical officers of health. It is probably a decided understatement to write that well over 50 per cent. of definite outbreaks are never recorded in available literature.

TUBERCULOSIS

This in man is the most important disease derived from the cow. Its incidence in human beings depends upon its prevalence in bovines, and the problem of its control is associated with the distribution of tuberculosis in bovines and particularly in cows.

No complete figures are available of the extent of bovine tuberculosis in Great Britain, but three sources of information have been utilized. One, that of animals killed under the Tuberculosis Order, 1913, is very unreliable and much affected by the extent to which the Order is enforced. It merely supplies corroborative evidence. The results of abattoir findings as to the number of animals found tuberculous on slaughter are a valuable guide, but are somewhat selective, as those slaughtered include an undue percentage of older animals and also many of poor quality.

The only reliable index of the extent of bovine tuberculosis is from the results of systematic testing of cows and cattle with tuberculin. This does supply accurate figures as to the number of infected animals, but this method neither measures the extent of the infection nor the degree of infectivity to man. Unfortunately such findings are only available for selected herds, mainly in connexion with the attested herds scheme or for the formation of tuberculin tested herds. This involves a considerable selection of herds, since those anticipated to

have a low incidence of tuberculosis are more liable to be selected. Again, the figures are usually given for cattle and not for dairy cows, the essential item from the human standpoint. These limitations must be borne in mind in assessing the results, but the findings are of great value. Ritchie and Stamp (1948) report that up to the end of October, 1947, 29,552 herds comprising 1,171,822 cattle have qualified for the attested herds register. This represents 13·4 per cent. of the cattle in Great Britain, i.e. in Scotland 30·1 per cent., Wales 24·5 per cent., England 7·9 per cent. When the results are distributed into counties they show very wide variations in incidence. Francis (1947) in his valuable book, *Bovine Tuberculosis*, gives the figures for each county for cattle (not cows) using the survey figures for 1938. For English counties those with percentages under 10 include Cumberland (7·0), Devon (5·6), Durham (4·5), Yorkshire (7·2–9·3), Hereford (5·1); over 30 per cent. positive were found in Cheshire (41·6), Derbyshire (32·2), Kent (37·0), and Nottinghamshire (31·2). Welsh counties ranged from 1·8 to 49·6 per cent. and Scottish counties from 2·6 to 66·3. These figures give the percentages for England as 14·1, for Wales 4·1, and for Scotland 13·0.

Abattoir figures are, in general, in accord with these findings. The *Veterinary Record* (1948) in an unsigned article gives an interesting series of maps showing the relative incidence in each county based upon this survey and other data.

Ritchie (1948) has made a more recent (1945) estimate and considers the incidence of "reactors," i.e. those positive to tuberculin tests, in cattle in England to be about 20 per cent., with 17-18 per cent. for Great Britain. He considers that this represents 30 to 35 per cent. of infection among cows.

These figures show that the incidence of tuberculosis in cows in Great Britain is very high and varies markedly from county to county. Also it may vary widely within the one county. For example Wilson (1937) in Herefordshire found percentages of 5 to 60 in different districts. The considerable number of herds freed from tuberculosis naturally reduces the percentage of "reactors" but as yet the proportion to the total cow population is too small to affect materially the overall figure. Allowing for the selective nature of these testings, it can be stated reliably that today about 35 per cent. of dairy cows are affected with tuberculosis in Great Britain. This is a reduction on the figure of 40 per cent. widely accepted for conditions existing a decade or so ago and is caused by the material progress being made in eradication.

All reactor cows are potential spreaders of tuberculosis to other animals, but their liability to be excretors of tubercle bacilli into the milk essentially depends upon the organs affected with tuberculosis. Tuberculosis of the udder is undoubtedly responsible for the heaviest infection of milk with tubercle bacilli. Savage in 1929 discussed the incidence and concluded that most of the figures advanced were too high, and estimated clinically detectable udder tuberculosis as 0·2 to 0·3 per cent. in unselected herds with a figure probably twice as high if all lesions demonstrable by pathological examination were included. The figures of Torrance (1936-37) support this estimate, as out of a cow population of 24,500 the use of biological tests, clinical examination, and pathological post-mortem examinations yielded 0·59 per cent. of udder tuberculosis. With 35 per cent. of reactors this figure suggests that 1·7 per cent. of all reactors suffer from tuberculosis of the udder and are especially liable to spread infection to man. Tubercle bacilli may get into the milk from tuberculous cows without udder lesions, as Griffith and Griffith (1911) demonstrated to the Royal Commission on Tuberculosis, but this is rare; also tubercle bacilli are excreted in dung, particularly from cows with intestinal tuberculosis, and so may get into the milk supply. Stamp (1943) gives a good account of the pathological lesions in udder tuberculosis and evidence of tubercular foci not visible to the naked eye.

The varied incidence of tuberculosis in dairy cattle in different parts of the country is reflected in a wide range of findings of tubercle bacilli in milk. Percentages ranging from 3 to 15 or even higher are recorded. When bulked milk is examined the percentage is very high, usually 80 to 100. For example, in Somerset milk samples collected from undesignated individual herds show over a period of 15 years ending in 1948 a percentage positive always between 2·0 and 3·0, except for 1940 and 1941 when they were 4·4 and 4·3.

One of the most comprehensive investigations was undertaken in Scotland (1933). The positive incidence in raw milk in churns for the four main cities was roughly 10 per cent., with Glasgow highest (13·4 per cent.) and Dundee lowest (6·6 per cent.). The incidence of infection increased with the volume of milk from which the sample was collected. Untreated retailed milk in the same four towns was 11·76 per cent. positive. Raw milk transported in tanks was 37·5 per cent. positive.

The proportion of human tuberculosis of bovine origin was largely guess work until bacteriologists were able to differentiate the type of tubercle bacillus

TABLE I

ANALYSIS OF DEATHS DUE TO BOVINE TUBERCULOSIS IN THE YEAR 1931

Type of Tuberculosis	Deaths under 5 years	Percentage Bovine	Calculated Deaths of Bovine Origin	Deaths over 5 years	Percentage Bovine	Calculated Deaths of Bovine Origin	All Ages: Calculated Bovine Deaths
Respiratory ..	303	0	0	27,355	1	294	294
Nervous	1,286	34	437	1,312	20	262	699
Abdominal ..	291	80	235	235	33	267	502
Generalized ..	327	25	82	971	9	87	169
Other varieties ..	55	20	11	1,115	5	56	67
All kinds ..	2,262	33	765	33,553	2·9	966	1,731

present in individual human cases. When a sufficient number of examinations of suitable material was available it was possible to determine with considerable accuracy the proportion of human tuberculosis due to the bovine tubercle bacillus. Savage (1929), using the official statistics of deaths from each variety of tuberculosis first made this calculation in 1927. A special tuberculosis committee of the People's League of Health reinvestigated the problem in 1931 and produced very similar figures. Savage brought his figures up to date in his Mitchell Lecture (1933) and Table I is taken from that lecture.

This and similar estimates have been accepted by all authorities but it is important to consider how far they are reasonably accurate for 1948. On the one hand tuberculosis as a whole has shown a steady decline, apart from a temporary war increase. We do not know whether that small proportion due to the bovine bacillus has declined proportionately, but in view of the great increase in the heat-treatment of milk in our large towns a considerable decline is likely to have resulted. On the other hand, in Table I there is one considerable miscalculation brought out by recent investigations. Over recent years much evidence has been adduced that the percentage of bovine infections in respiratory tuberculosis is much higher than was originally accepted. This is particularly the case in Scotland where Munro (1939) found 50 cases of bovine infections out of 1,061 respiratory tuberculosis cases, a percentage of 4·7, at Glenlomond Sanatorium, and quotes Griffith's findings at two other Scottish sanatoria of 24 bovine out of 534 cases, a percentage of 4·5. Cutbill and Lynn (1944) in Cheshire found 48 bovine infections in 2,101 pulmonary tuberculosis cases, collected over a period of 15 years, a percentage of 2·3.

In their most recent paper Griffith and Munro (1944) have summarized the figures up to 1944 including their own work and that of Cumming, Lynn and Cutbill, Paget, and others. These are set out briefly in Table II.

The respiratory tuberculosis cases of bovine origin were distributed unequally in the different regions and, in general, with gradually decreasing frequency from the north of Scotland to the south of England.

Lange (1932) of Berlin took the view that most of these pulmonary cases associated with the bovine strain were attributable to infection in connexion with their employment. Undoubtedly some of the infections occur in this way as direct infections from tuberculous cattle, but Griffith and Munro found that for the great majority of the cases the alimentary canal was the portal of entry with infected milk as the probable source of infection. A very few cases were due to infection from a human case infected with the bovine tubercle bacillus.

Human pulmonary tuberculosis due to the bovine bacillus is met with in other countries. For example Knudsen (1947) in Denmark, out of 113 cases isolated 13 (11·5 per cent.) of bovine type, and Ruys in the Netherlands obtained high figures (see Table V).

For calculation purposes it is safe to assume that at least 2 per cent. of cases of respiratory tuberculosis are due to the bovine bacillus. This would add 294 deaths to Table I, increasing the total estimated

TABLE II

INCIDENCE OF BOVINE TUBERCULOSIS

Country	No. of Cases Investigated	Cases due to Bovine Bacillus	Percentage Bovine
Scotland ..	2,769	160	5·7
England, Wales Eire ..	4,194	81	1·9

deaths to 2,025. Allowing for the undoubted decline in incidence, about 1,500 deaths a year would be a reasonable estimate for the present time. To these must be added the several thousand cases a year due to bovine infections not resulting in death.

The higher incidence of tuberculosis, in all its forms, of bovine origin in Scotland is well known, but recent work has shown a definite relationship in incidence between town and country. A few examples may be quoted.

Lethem (1946), selecting abdominal tuberculosis in children under five years as usually due to the bovine strain, gives the following figures (Table III).

TABLE III

COMPARISON OF INCIDENCE BETWEEN TOWN AND COUNTRY

Area	Death Rate per Million			
	1921	1930	1938	1944
London: Administrative County	136	24	12	6
Combined county boroughs	437	157	63	35
Combined urban districts	366	134	77	42
Combined rural districts ..	252	92	63	60

This table illustrates the much higher rates in rural districts, as well as the striking reductions since 1921. By 1930 the proportion of heat-treated milk in the Administrative County of London had risen to about 90 per cent., whereas in 1939 it was 98 per cent., the corresponding figures in county boroughs being 35 per cent. and 60 per cent.

Blacklock (1947), as the result of detailed bacteriological and pathological examinations of children under 13 years infected with the tubercle bacillus, found the bovine percentages to be: primary lung lesions 3·6, abdominal 80·8 (1914-32) and 64·7 (1943-44), cervical glands 70·2 (1914-35) and 67·5 (1943-44).

The distribution figures are interesting.

TABLE IV

DISTRIBUTION

Area	Tuberculous Meningitis (% Bovine)	Surgical Tuberculosis (% Bovine)	Heat-treatment of Milk Supply
Glasgow ..	2·3	8·8	95% heat-treated
Large boroughs in the Clyde Valley	8·2	13·6	Considerable but much less than in Glasgow
Rural areas in the western counties	10·9	44·4	Practically no heat-treatment

Cervical adenitis cases of bovine origin were 77·2 per cent. bovine in country areas compared with 46·8 per cent. in urban areas.

Price (1938) in Canada examined a series of 500 tuberculous children under 14 years of age over a period of 13 years. Of these, 9 per cent. of cases of extra-pulmonary tuberculosis were of bovine origin. None of the cases derived from Toronto, a large city with 100 per cent. pasteurization of milk. All the cases of bovine infection came from Ontario outside Toronto, and the history invariably revealed that the child had been fed with raw milk. From 200 samples of raw milk coming into the city to be pasteurized, 26 per cent. yielded living tubercle bacilli.

Ruys (1939), including her own findings and those from other Dutch investigators, gives the following figures for 1933-38.

Here the difference between town and country is present but not marked, for it was only since 1940 that pasteurization was extensively used in Amsterdam and elsewhere. For 1933-39 the bovine

TABLE V

INCIDENCE OF BOVINE TUBERCULOSIS IN THE NETHERLANDS (1933-38)

	Pulmonary Tuberculosis				All Other Forms of Tuberculosis			
	All Ages		0-15 years		All Ages		0-15 years	
	No. Examined	Bovine (%)	No. Examined	Bovine (%)	No. Examined	Bovine (%)	No. Examined	Bovine (%)
Towns over 100,000	699	4·3	282	9·2	229	20·5	71	21·1
Rest of the country	619	6·8	116	9·4	260	25·4	41	43·9

percentage for pulmonary tuberculosis in Amsterdam was 9·6, for 1940-44 only 1·8, with a corresponding decline for other types.

Greater London, Scotland, Canada, the Netherlands, all yield the same picture of a striking reduction of bovine infection with the increased ratio of pasteurized to unpasteurized milk.

Tuberculosis of bovine origin is becoming mainly a rural and small urban district problem.

Brucellosis in Man (Undulant Fever)

Of the three brucella types *Br. melitensis*, *Br. abortus* and *Br. suis*, the only one which affects dairy cattle in Great Britain is the *abortus* strain, and this is the cause of contagious abortion in cows and of undulant fever in man. Human infections in Great Britain with the other two strains are rare indeed. They occasionally occur in cows, and Duke (1940) in a bulk sample from an accredited herd of 76 cows isolated *Br. melitensis* and subsequently from three of the cows. No goats were kept on the farm. *Br. suis* seems to be rather more prevalent in the U.S.A. Jordan, Borts *et al.* (1943) describe an outbreak of human brucellosis of 77 cases in Marcus (Iowa) due to *Br. suis* and mention an earlier outbreak in 1933 due to the same type. In Iowa, a cattle and hog raising state, the official notification rate of human brucellosis over a 12-year period was 6·25 per 100,000.

Contagious abortion is widely prevalent in Great Britain and its extent can be judged from examinations of milk. These show that some 20 to 30 per cent. of herd milk samples contain living *Br. abortus* organisms but the percentage varies widely in different districts as shown by the following examples. Pullinger (1934) found 37 per cent. of mixed milk from single farms positive in Cheshire and only 19 per cent. positive in Somerset. Priestley (1934) using positive agglutination reactions as criteria records variable figures, but not less than 20 per cent. of cows showed antibodies specific to *Br. abortus* in their blood. Smith (1934), in the Aberdeen area, examined samples of milk from 183 single cows and 17 (9·2 per cent.) yielded *Br. abortus*. Menton (1940) examined over 1,000 milk samples from the Midlands and 34·5 per cent. showed positive agglutinins, but living *Br. abortus* was only isolated from 29·1 per cent., whereas a further 9·1 per cent. of samples contained *Br. abortus* but were devoid of agglutinins. Jones (1943) examined 314 samples of milk in the Liverpool area (1933-34) and 15·3 per cent. contained *Br. abortus*, whereas of 408 samples from Kent 14·5 per cent. were positive.

The number of cases each year of undulant fever is not known, as many cases are not diagnosed while the disease is not a notifiable one, but it is possible to give an estimate. The most reliable basis is from large-scale serum agglutination testings. These have not been carried out systematically, particularly in rural areas where a higher incidence may be anticipated. Of 3,175 sera examined from various sources 101 were positive, a percentage of 3·18, which corresponds to 440 new cases a year (Topley and Wilson, 1946). In addition there are many cases of latent infection, with positive serum agglutination below the significant figure of 1·80+, infections associated with the comparatively low pathogenicity of *Br. abortus* to man. Wilson's (1932) estimate of about 500 new cases a year is probably near the mark.

Dalrymple-Champneys (1948) has collected records of some 900 cases of undulant fever and his paper gives a good account of its epidemiology. The case mortality in his series was only 2·3 per cent., but the disease often causes prolonged incapacity. The majority of recorded cases are single infections but a number of small outbreaks have been recorded in schools, such as those described by Elkington *et al.* (1940), by Cruickshank and Stevenson (1942), and the small outbreak of four cases at a school at North Molton (1942).

The discrepancy between the high incidence of contagious abortion in cows and the comparatively few cases of undulant fever in man is partly accounted for by the probability of numerous minimal infections with no definite symptoms but causing immunity, and partly to the low virulence of *Br. abortus* to man.

Although human infection is usually due to the consumption of infected raw milk an unknown proportion is spread by direct contact with infected animals amongst farm workers, veterinary surgeons, and men engaged in animal slaughtering. Smith (1934) examined the sera of 106 men associated with meat and slaughtering. He found 16·1 per cent. of sera from slaughtermen and 10·5 per cent. of sera from men working in allied trades agglutinated *Br. abortus* 1: 25 or more, compared with 6·8 per cent. from unselected cases. There was no incidence of undulant fever but some evidence of latent infections. This is in accord with other studies, but some investigators consider a material proportion of cases are contact infections.

Streptococcal Infections

These outbreaks fall into two of the three groups mentioned at the beginning of this paper, namely that due to infection of the milk from human strains implanted in the udder of the cow or to specific

contamination of the milk after it leaves the cow. The human diseases are outbreaks of septic sore throat, scarlet fever and, very rarely, streptococcal food poisoning.

Mastitis is a prevalent disease in cows, but the organism mainly responsible is *Str. agalactiae* and this type is harmless to man, as Savage (1911) demonstrated in 1906-11. He further showed that when a cow with mastitis is responsible for a human outbreak of sore throat it is because the udder has been invaded and infected with a *Str. pyogenes* strain of human origin. This view is now fully accepted and, using the typing methods of Lancefield and Griffith, it can be regularly demonstrated.

The bovine types, including *Str. agalactiae*, may or may not be haemolytic but are nearly all Type B. Isaac and Nussbaum (1940), for example, examined 93 milk samples for haemolytic streptococci: Type A strains were absent and Type B were over 80 per cent. The pathogenic strains are about 90 per cent. Type A and this type includes all those responsible for sore throat outbreaks. They belong, however, to a number of Griffith types.

These pathogenic streptococci do not readily establish themselves in the bovine udder, but this is facilitated if there are udder or teat lesions. This accounts for the comparative rarity of such outbreaks in Great Britain, although they seem to be more prevalent in the U.S.A. Examples of widespread outbreaks are the Colchester outbreak in 1905 with over 600 cases and the Hove-Brighton outbreak of 1929-30 with more than 1,000 cases and over 60 deaths.

Until the last 30 years or so, milk-spread outbreaks of scarlet fever were ascribed to direct infection of the milk from a case or carrier. Although this is the method of spread in some instances, the evidence strongly suggests that the majority are due to udder infection with the pathogenic streptococcus, exactly as for sore throat outbreaks. The evidence is along four lines.

Various investigators have shown that *Str. pyogenes* strains grow with difficulty and slowly in ordinary milk. Jones demonstrated this as early as 1928 and ascribed it to a definite inhibitory substance in milk, destroyed when milk is boiled. Davis (1914) showed the same finding for milk at 26° C. using a strain directly isolated from a sore throat outbreak at Chicago and confirmed that with sterile milk considerable multiplication occurred. Pullinger and Kemp (1937) found similar failure to multiply at 18-20° C. in unsterilized milk and also that the initial degree of contamination with *Str. pyogenes* had no influence upon their inability to multiply.

Epidemiologically it is difficult to account for scarlet fever outbreaks which have a considerable duration unless there is some nidus which continues to infect the milk.

In most recent outbreaks the cases consist of a mixture of clinical scarlet fever with rash and sore throat and those without rash, so we should expect causation to be similar to outbreaks of infective sore throat and the relationship of these outbreaks to infected cows is proved to the hilt.

In many of the more recent scarlet fever outbreaks it has been possible to prove the association of the outbreak with a cow with an udder infected with a streptococcus of the same type as the human case.

The 1936 Doncaster (Watson, 1937) outbreak was a mixture of 135 sore throat cases with rash (scarlet fever) and 229 similar cases without rash, and it was spread over 11 days. The source of infection was a cow with an indurated teat excreting the same streptococcus, Type 2, as was recovered from the patients. The 1939 Elgin (Douglas *et al.*, 1942) outbreak with 214 cases, some with, some without rash, was due to a Type 3 strain recovered both from patients and the infected cow. The earlier Chelmsford (Camps and Wood, 1936) outbreak of 1935 had the same clinical features and was due to Type 2. It lasted 11 days only and probably had the same causation but was not proved, as unfortunately only one cow was bacteriologically investigated.

Benedixen and Minett (1938) kept the Doncaster infected cow and a cow responsible for a similar outbreak at Morkov (Denmark) under observation. The Danish cow continued to excrete the *Str. pyogenes* strain from the affected quarter for at least 13 months and the Doncaster cow for about six weeks.

In the U.S.A. this association with bovine infection is more widely recognized. Dublin *et al.* (1943) record that there were 57 outbreaks of scarlet fever and sore throat in New York (excluding New York City). The strain was typed in some outbreaks and three were Type 3, one Type 2, and the rest other types. The majority were associated with an infected udder.

There is no evidence that the type of clinical case has any relationship to the streptococcus type, although Evans (1946) from a study of 23 such strains suggests that this is so.

So far it has been a rare occurrence, but severe septic infection may be the essential feature as in the interesting little outbreak described by Golledge (1932) with four cases of whom two died. They were infected from two cows suffering from mastitis, and the strain recovered from cows and patients was *Str. pyogenes* Type 1.

When outbreaks result from direct infection of the milk, without cow intervention, they are usually explosive and limited in duration. The mixed scarlet fever and sore throat outbreak at Vejle in 1937 (Henningsen and Ernst, 1939) is a good example. About two-thirds of the cases were spread over four days, and the authors think that infection of the milk may have been confined to a single day. No evidence of cow infection was found, but the source was a milkmaid with purulent otitis media following unrecognized scarlet fever. Both from her and from many other cases *Str. pyogenes* Group A Type 3 was recovered.

Enteric Infections

Typhoid fever is usually water-borne, but sometimes the actual vehicle is milk contaminated with water containing typhoid bacilli used for washing utensils or for other purposes. Most milk-spread outbreaks are, however, due to direct infection of milk from a milk handler who is either an acute case or, more frequently, a chronic carrier.

Milk-spread typhoid outbreaks may be very extensive, such as the 1936 outbreak in Bournemouth and Poole (Shaw, 1937). This outbreak attacked at least 718 persons and caused about 70 deaths; it is also interesting since it was suggested that infection might have been transmitted through dung following systematic infection of a cow from drinking contaminated water. No evidence in support was adduced and direct experiments by Scott and Minett (1947) with a cow fed with virulent typhoid bacilli were entirely negative.

Improved methods for the isolation of *B. typhosus* and new methods which divide the bacillus in types have shown how wide may be the ramifications of milk-spread typhoid fever and also that comparatively few persons may be infected. Bradley (1943) supplies a particularly interesting illustration of 21 typhoid cases spread over two years (1941-42) and located in four different counties. This involved many milk distributors, and all the cases were traced to a typhoid carrier who infected the milk on a farm in Wiltshire 100 miles away. This carrier and all the cases were infected with the rather rare D4 type.

The important fact that these milk-spread outbreaks are by no means either always explosive in character or extensive is well illustrated by the little outbreak at Sedgley reported by Bekenn and Edwards (1944). Over a period of 10 months there were six apparently disconnected cases of typhoid fever, but all milk-spread and traced to one farm where an old woman of 78 years was a chronic typhoid carrier.

Paratyphoid fever is rarely water-spread but milk is a common vehicle. In a study of 40 recent outbreaks Savage (1942) records that eight were milk-spread, whereas 16 were from cream, fresh or mostly artificial, or cream cakes. The source of infection of the milk is a temporary or chronic paratyphoid bacillus carrier.

There is no doubt that not only have single cases and outbreaks of dysentery been more numerous in recent years but there has been a proportionate increase in those milk-spread. Their severity mainly depends upon the dysentery strain responsible. Those due to the Flexner type are often extensive and severe with a high mortality as in the Aberdeen (Kinloch, 1923) outbreak of 1919 with over 1,000 cases and 72 deaths. The most frequent causative strain in recent outbreaks has been the Sonne type, usually with a very low mortality. Outbreaks recorded over the past seven years, due to the Sonne type, are set out in Table VI. Frequently it was not possible to ascertain the exact source of infection.

Dysentery bacilli do not appear to grow easily or even to survive in raw milk, but Steuer (1941) records periods of survival of from one to 15 days at room temperature.

TABLE VI

Analysis of Outbreaks of Dysentery (Sonne Type) over Seven Years

Year	Place	No. of Cases	Deaths	Authors of Report
1942	Cardiff	27	0	Hobbs and Allison
1942	Cumberland ..	43	0	Faulds
1942	Penrith	178+	0	Faulds
1943	Silloth	124	0	Faulds
1944	Dorset	57	0	O'Keefe and Cooper
1945	Cheeping Wycombe	76	0	Muir and Vollum
1945	N.E. Scotland ..	250	0	Rae and Smith
1947	Scotland	84+	0	Henderson, Michie, Rae, and Smith

Diphtheria

Diphtheria outbreaks spread through milk are comparatively infrequent: nevertheless, as shown in the figures given, there have been a good many of them. The milk itself is directly infected from an actual case or a carrier of diphtheria bacilli, and usually such a person has been a milker. In some of the recent outbreaks, such as the East Yorkshire outbreak of 1941 described by Goldie and Maddock (1943), it has been possible to isolate *C. diphtheriae* from the infected milk. This outbreak is interesting for the small number of cases compared with those at risk. This has occurred in some other outbreaks and the factor of dilution is important.

An interesting feature in a few outbreaks has been the association of the human cases with sores shown to be infected with diphtheria bacilli on the teats of some of the cows. This was first noted by Dean and Todd in 1902 and later instances are recorded by Ashby (1906), Robertson (1911), Macdonald (1913), and McSweeney and Morgan in 1928. They are surface infections and the bacilli do not invade the udder structure. Their possible occurrence is of epidemiological importance, since they may be responsible for prolonging infection and may explain continuance of cases without any explosive outbreak.

It is doubtful if diphtheria bacilli multiply readily in raw mixed milk. In a few experiments made by Goldie and Maddock (1943) the bacilli remained about the same number for four to six hours at room temperature but then gradually diminished.

Food Poisoning

Milk may be the vehicle for two varieties of food poisoning, i.e. the staphylococcus toxin type and that due to Salmonella organisms.

Staphylococcus Food Poisoning.—Only a small minority of staphylococcus strains produce the special toxin (enterotoxin) which is responsible for symptoms of food poisoning. All are coagulase-positive, most are of *aureus* type, but only a proportion of strains with these properties produce enterotoxin and so cause food poisoning.

Many foods can act as a vehicle and milk is not a very common one, although it was responsible for the first recorded outbreak of this nature. A human carrier is the common source of infection but the possibility of staphylococci from the cow's udder being a source has been a good deal discussed. Staphylococci are fairly common in milk drawn direct from the cow's udder, healthy or diseased. Minett (1938) isolated 38 strains of *Staph. aureus* from the bovine udder, seven being from cows with acute mastitis, eight with chronic mastitis, and the rest from cows with normal udders. Enterotoxin was reported, having been demonstrated with four, five, and seven strains respectively from these three sources. This was based on the so-called " kitten test," but results were negative or very indefinite when feeding experiments were conducted with human volunteers. Dolman (1941 and 1943), who has done so much valuable work on this variety of food poisoning, considers that endogenous infection of milk is an important source of infection. Out of 74 samples of raw milk examined he claims that 64 contained toxigenic staphylococci. The difficulty of accepting these findings is that they are based upon the " kitten intraperitoneal test " which has been severely criticized as unreliable. The only completely satisfactory test is human feeding experiments and these have not been used. The evidence suggests that only exceptional strains from milk produce enterotoxin and that this is not a common source of infection.

An important fact in these outbreaks, with any food as a vehicle, is the significance of the time-temperature factor after infection to allow sufficient enterotoxin to be produced to set up food poisoning.

Salmonella Food Poisoning.—Outbreaks of this type are not infrequent, either from the direct consumption of milk, or of its products. They may be extensive and are explosive in character. Of earlier outbreaks may be mentioned Newcastle-upon-Tyne (1913), 523 cases, 0 deaths; Withnell and Chorley (1914), 317 cases, 2 deaths; Newcastle-under-Lyme (1914), 468 cases, 2 deaths; Glasgow (1914), 370 cases, 0 deaths; Aberdeen (1925), 497 cases, 1 death. It is noticeable that in these milk-spread outbreaks the mortality is conspicuously low.

For most of these outbreaks the source of infection was a diseased cow. For instance, in the 1925 Aberdeen outbreak infection was traced back to a cow with an indurated udder, which developed septicaemia from whom *Sal. enteritidis* was isolated from udder and flesh. The outbreak of 1947 from the same area was also traced to a cow which died from *Sal. enteritidis dublin*, and this strain was isolated from all organs. Probably most of these outbreaks were due to the Dublin sub-variety but the differentiation was unknown for the earlier outbreaks. The very considerable extent to which *Sal. enteritidis dublin* is a cause of bovine infections has been only fully recognized in recent years and merits serious recognition. Boswell and Lovell as early as 1931 described three outbreaks in calves due to *Sal. dublin.* Craig, Davies, and Massey (1941) showed that this strain was responsible for a number of cases in a herd in Cheshire, the cases

cropping up over a number of years. They found no carrier cases. John (1946) gives particulars of four cases and found that all the cows that recovered became carriers and continued to excrete the bacillus for long periods. Bythell (1946) described outbreaks of 12 cases in all, one in Shropshire, and one in Staffordshire in 1945, when all the cases were fatal. Grunsell and Osborne (1948) found sporadic cases due to *Sal. dublin* on five farms in the Glastonbury (Somerset) area. The organism was isolated both from the faeces and the milk of infected animals. Field (1948) diagnosed bovine salmonellosis on 70 farms in west Wales during the two years 1946-47. Most of these resulted during the warmer (June-October) months of the year. In all but four the strain responsible was *Sal. dublin*. In his cases most of the animals which recovered remained carriers, and were responsible for a number of subsequent infections, mostly to calves. He also showed that this strain can survive for long periods in faeces and also in water.

With such widespread herd infections, it is surprising that more human outbreaks from milk have not been recorded. One typical outbreak due to *Sal. dublin* occurred in a Wiltshire school (Conybeare and Thornton, 1938) with over 100 children affected with gastro-enteritis, the vehicle being the school milk consumed raw. This strain was isolated from the patients, from the milk, and from the dung of one of the cows. The Knaresborough outbreak of 1944 (Sutherland and Berger) with at least 162 cases had exactly the same origin. In this outbreak the infective cow was transferred to the Weybridge laboratory for later investigation, and Rankin and Slavin (1947) found that the bacillus continued to be excreted in very large numbers (at one time 2,580 per g. in the excreta), but milk samples were negative.

Other Milk-spread Diseases

Foot and mouth disease is an infection of cows which may be transmitted to man, but is decidedly rare. Whether infection is always direct or can be transmitted through milk is uncertain, but Dlugosz (1943) who describes two clinical cases without, however, any information as to method of infection, mentions that French scientists have experimentally proved infection through drinking milk. (The original reference cannot be traced.) Anthrax readily affects cows, but the animals die so quickly that spread through milk is a very unlikely contingency. In 1910 there was an extensive outbreak of anthrax in Chicago, involving 20 farms, and 87 cows died, but no persons were infected through milk.

The spread of epidemic poliomyelitis through infected milk has been advanced, but a study of the evidence shows that it is inconclusive and at present a verdict of "not proven" seems the correct attitude. Those interested will find evidence in favour of transmission in individual outbreaks in papers by Jubb (1915), Watts (1927), Aycock (1927), and Goldstein *et al.* (1946).

Milk Products

The only milk products which require consideration are cream, butter, and cheese.

Cream.—Cream being merely part of milk can convey all the diseases spread by milk if it is unpasteurized. Tubercle bacilli have frequently been found in raw cream. For example, Pullinger (1942) found tubercle bacilli in 16 out of 31 samples and *Br. abortus* in 11 out of the 31.

Cream has been responsible for several outbreaks of paratyphoid fever (Savage, 1942) but less frequently than artificial cream. Typhoid fever outbreaks due to cream seem to be very infrequent although the typhoid bacillus survives for a good many days in cream, and Brock (1903) found survival for at least 10 days. It has been the vehicle in a good many outbreaks of staphylococcus food poisoning, but is rarely a vehicle in salmonella poisoning.

Butter.—Theoretically tubercle bacilli should not be infrequent in butter, but most investigations show a very low prevalence. For instance Edington (1934) out of 40 samples (14 from Denmark, 25 from New Zealand, and one from Australia) found it in only one Danish sample. Pullinger (1935) failed to detect it in 39 samples, all except nine home produced. *Br. abortus* was also absent. Smith (1934) examined 51 samples of butter prepared on individual farms and five showed tubercle bacilli but no *Br. abortus*, but another series of 15 samples of English dairy butter, five of colonial, and eight from foreign sources were free from both organisms.

Pullinger and Kemp (1938) have shown that various salmonella strains (including *B. paratyphosus B*) can survive in both salted and unsalted butter for at least two months. Theoretically, therefore, they could be responsible for outbreaks of salmonella food poisoning, but the writer knows of no reported outbreaks either of this condition or of enteric outbreaks in Great Britain. On the other hand Studeny (1947) in Austria has reported three outbreaks of paratyphoid fever with butter as the vehicle. The first outbreak at Graz in 1940 of 143 cases was traced to butter supplied from a central factory and due to a paratyphoid carrier on the staff. Although a new factory was erected, a second outbreak was traced to a carrier on the staff of this

factory also; 365 cases were spread by milk and butter. The third outbreak of 35 cases was from farmhouse butter derived from different hill farmers.

Cheese.—Cheese has been responsible for numerous outbreaks, and the literature is so extensive that space will only permit of a general summary. Direct experiments show that bovine tubercle bacilli can survive in cheese for many months, and Pullinger (1935) has demonstrated it on four out of 63 samples of cheese from various sources. No examples of human infection have been recorded, but such evidence is almost impossible to establish.

Br. abortus can rarely be found in cheese, and Smith (1934) failed to isolate it from 63 samples from various sources. On the other hand direct experiments have demonstrated its survival after inoculation for periods up to 60 days, but rarely beyond 50 days. Gilman, Dahlberg, and Marguardt (1946) specially studied survival and concluded that an ageing period of 60 days is adequate for safety.

The likelihood of infected cheese spreading undulant fever seems remote, but this does not seem true for infections by *Br. melitensis*, for Celli (1944) has reported a small outbreak in Italy, and Veloppe and Jaubert (1925) an outbreak of 14 cases and two deaths from cheese eaten freshly made.

For well over 60 years cheese has been reported as responsible for outbreaks of food poisoning. Under the influence of the investigations by Vaughan (1903) these were ascribed to tyro-toxicon, a ptomaine-like body, but this conception has long been abandoned. Cheese continues to be a vehicle of food poisoning due either to salmonella strains or of enterotoxin type. Savage and White (1925) in their report of 100 outbreaks of food poisoning found that in eight the vehicle was cheese. Experimentally they showed survival of two salmonella strains in cheese up to 30 days but not beyond. Much more recently Tucker *et al.* (1946) held artificially inoculated cheese at 43-48° C. and found survival of *Salm. typhi murium* up to 302 days. This organism was responsible for an outbreak of 384 cases and the vehicle was cheddar cheese sold soon after manufacture.

Many food poisoning outbreaks are recorded by Berberean (1946) in Syria and the Lebanon due to cheese, usually eaten as green cheese and prepared locally. Most are ascribed to *Staphylococcus aureus*. Erkmen (1941) refers to 21 outbreaks of food poisoning type (1937-39) in Turkey from cheese.

The writer is unaware of any recent outbreaks of typhoid fever or paratyphoid fever in Great Britain with cheese as the vehicle, but over recent years a considerable number has been reported in the U.S.A. and particularly in Canada. Some have been extensive. For example, with six outbreaks in Canada the known cases have been 8, 21, 100, 29, 83, and 40, respectively, and of two in the U.S.A., 225 and 77. Most of the cases have one feature in common, that the cheese has been eaten as " green cheese," i.e. before an adequate period of ripening has been allowed to elapse. One report will serve as an example. Menzies (1944) describes three different cheese-borne outbreaks of typhoid fever in Alberta, in 1936, 1938, and 1944. The cases in the 1944 outbreaks numbered 83, with seven deaths all spread over a wide area with only cheese as a common vehicle. In all the cases fresh green Cheddar cheese made from raw milk was consumed. The source was traced to a typhoid carrier working in the production factory. These outbreaks raise the problem of the survival of typhoid bacilli in cheese, and Campbell and Gibbard (1944) studied this with three strains. Survival varied with the temperature of holding. At 40-42° F. survival was 182-336 days whereas at 58-60° F. it was from 35 to 196 days.

Canada, and at least seven states in the U.S.A., now require cheese to be made either from pasteurized milk or cream or to be held in storage from 60 to 120 days in the U.S.A. and for 90 days in Canada.

References

Aycock, W. L. (1927). *Amer. J. Hyg.*, **7**, 791.

Bekenn, C. H. G., and Edwards, J. L. (1944). *Mon. Bull. Min. Hlth*, **3**, 60.

Bendixen, H. C., and Minett, F. C. (1938). *J. Hyg., Camb.*, **38**, 374.

Berberean, D. A. (1946). *J. Palestine Arab med. Ass.*, **1**, 30.

Blacklock, J. W. S. (1947). *Brit. med. J.*, **1**, 707.

Bradley, W. H. (1943). *Ibid.*, **1**, 438.

Bruck, C. (1903). *Dtsch. med. Wschr.*, **29**, 460.

(1942). *Bull. Emergency publ. Health Lab. Service*, January, p. 12.

Bythell, D. W. P. (1946). *Vet. Rec.*, **58**, 425.

Campbell, A. G., and Gibbard, J. (1944). *Canad. J. publ. Hlth*, **35**, 158.

Camps, F. E., and Wood, J. L. M. (1936). *Lancet*, **2**, 756.

Cilli, V. (1944). Dairy Sci. Abstract, **6**, 39.

Cruickshank, J. C., and Stevenson, G. A. (1942). *Brit. med. J.*, **1**, 522.

Conybeare, E. T., and Thornton, L. H. D. (1938). *Ministry of Health Public Health Report*, No. 82. London.

Craig, J. F., Davies, G. O., and Massey, Kathleen M. (1941). *Vet. J.*, **97**, 145.

Cutbill, L. J., and Lynn, A. (1944). *Brit. med. J.*, **1**, 283.

Dalrymple-Champneys, W. (1948). *Publ. Hlth, Lond.*, **61**, 239.

Davis, D. J. (1914). *J. infect. Dis.*, **15**, 379.

Dlugosz, H. (1943). *Brit. med. J.*, **1**, 189.

Dolman, C. E. (1941). *Canad. J. publ. Hlth*, **32,** 183.
——— (1943). *Ibid.*, **34,** 97, 205.
Douglas, R., Smith, J., Sutherland, I. N., and Watson, R. J. P. (1942). *J. Hyg., Camb.*, **41,** 543.
Duke, F. W. (1940). *Lancet*, **2,** 517.
Dublin, T. D., Rogers, E. F. H., Perkins, J. E., and Graves, F. W. (1943). *Amer. J. publ. Hlth*, **33,** 157.
Edington, J. W. (1934). *Lancet*, **2,** 81.
Elkington, G. W., Wilson, G. S., Taylor, Joan, and Fulton, F. (1940). *Brit. med. J.*, **1,** 477.
Erkmen, R. (1941). *Türk. Z. Hyg. exp. Biol.* (German summary), **2,** 142.
Evans, Alice C. (1946). *J. infect. Dis.*, **78,** 18.
Field, H. I. (1948). *Vet. J.*, **104,** 323.
Francis, J. (1947). "Bovine Tuberculosis." London. Staples Press Ltd.
Gillman, H. L., Dahlberg, A. C., and Marquardt, J. C. (1946). *J. Dairy Sci.*, **29,** 71.
Goldie, W., and Maddock, E. C. G. (1943). *Lancet*, **1,** 285.
Goldstein, D. M., Hammon, W. M., and Viets, H. R. (1946). *J. Amer. med. Ass.*, **131,** 569.
Golledge, S. V. (1932). *Vet. Rec.*, **44,** 1499.
Griffith, A. S., and Griffith, F. (1911). Final Report of the Royal Commission on Tuberculosis, Appendix III. London.
———, and Munro, W. T. (1944). *J. Hyg., Camb.*, **43,** 229.
Grunsell, C. S., and Osborne, A. D. (1948). *Vet. Rec.*, **60,** 85.
Henningsen, E. J., and Ernst, J. (1939). *J. Hyg., Camb.*, **39,** 51.
Isaac, M. L., and Nussbaum, M. (1940). *Amer. J. publ. Hlth*, **30,** Suppt. 1.
John, F. V. (1946). *Vet. Rec.*, **58,** 211.
Jones, E. R. (1943). *J. Path. Bact.*, **55,** 357.
Jones, F. S. (1928). *J. exp. Med.*, **47,** 965.
Jordan, C. F., Borts, I. H., Harris, D. M., and Jennings, J. R. (1943). *Amer. J. publ. Hlth*, **33,** 773.
Jubb, G. (1915). *Lancet*, **1,** 67.
Kinloch, J. P. (1923). *J. Hyg., Camb.*, **21,** 451.
Knudsen, Margrethe (1947). *Ugeskr. Laeg.*, **109,** 93.
Lange, B. (1932). *Brit. med. J.*, **2,** 503.
Lethem, W. A. (1946). *Mon. Bull. Min. Hlth*, **5,** 80.
McSweeney, C. J., and Morgan, W. P. (1928) *Lancet*, **2,** 1201.
Menton, J. (1940). *Med. Offr*, **63,** 33.
Menzies, D. B. (1944). *Canad. J. publ. Hlth*, **35,** 431.
Minett, F. C. (1938). *J. Hyg., Camb.*, **38,** 623.
Munro, W. T. (1939). *Edinb. med. J.*, **46,** 165.
The People's League of Health (1932). Special Committee Report on a Survey of Tuberculosis of Bovine Origin in Great Britain. London.
Price, R. M. (1938). *Canad. J. publ. Hlth*, **29,** 251.
Priestley, F. W. (1934). *J. comp. Path. Therap.*, **47,** 181.
Pullinger, E. J. (1934). *Lancet*, **1,** 967.
——— (1935). *Ibid.*, **1,** 1342.
———, and Kemp, A. E. (1937). *J. Hyg., Camb.*, **37,** 527.
——— ——— (1938). *J. Hyg., Camb.*, **38,** 587.
Rankin, J. D., and Slavin, G. (1947). *Vet. Rec.*, **59,** 122.
(1934). Economic Advisory Council, Report of Committee on Cattle Diseases. Cmd. 4591. London.
Ritchie, J. N. (1948). *J. R. sanit. Inst.*, **68,** 503.
———, and Stamp, J. T. (1948). *Tubercle, Lond.*, **29,** 34, 61.
Ruys, A. Charlotte (1939). *Ibid.*, **20,** 556.
Savage, W. G. (1911). *Proc. R. Soc. Med.*, **4** (Epidem. Section), 73.
——— (1929). "The Prevention of Human Tuberculosis of Bovine Origin." Macmillan & Co., London.
——— (1933). *Brit. med. J.*, **2,** 905.
——— (1942). *J. Hyg., Camb.*, **42,** 393.
———, and White, P. B. (1925). *Spec. Rep. Ser. med. Res. Coun., Lond.*, No. 92.
Scott, W. M., and Minett, F. C. (1947). *J. Hyg., Camb.*, **45,** 159.
Shaw, W. V. (1937). *Ministry of Health Public Health Report*, No. 81. London.
Smith, J. (1934). *J. Hyg., Camb.*, **34,** 242.
——— (1934). *Ibid.*, **34,** 244.
Stamp, J. T. (1943). *J. comp. Path. Therap.*, **53,** 220.
Steuer, W. (1941). *Arch. Hyg., Berl.*, **126,** 233.
Studeny, O. (1947). *Wien. med. Wschr.*, **97,** 347.
Sutherland, P. L., and Berger, F. M. (1944). *Brit. med. J.*, **1,** 488.
Topley, W. W. C., and Wilson, G. S. (1946). Revised Wilson, G. S., and Miles, A. A. "Principles of Bacteriology and Immunity." Third Edition. Page 1698. London.
Torrance, H. L. (1936-37). Notts. County Council Report.
(1933). "Tuberculosis Infection in Milk." Report by the Department of Health for Scotland, *Spec. Rep. Ser. med. Res. Coun.*, No. 189. H.M.S.O., London.
Tucker, C. B., Cameron, G. M., Henderson, M. P., and Beyer, M. R. (1946). *J. Amer. med. Ass.*, **131,** 1119.
(1948). Annotation *Vet. Rec.*, **60,** 302.
Vaughan, V. C., and Novy, F. G. (1902). *Cellular Toxins*, Philadelphia.
Veloppe, and Jaubert, (1935). *Rev. Gen. de Med, Vet.*, **44,** 513.
Watson, R. (1937). *Brit. med. J.*, **1,** 1189.
Watts, A. W. (1927). *Ibid.*, **1,** 114.
Wilsdon, A. J. (1937), *Vet. J.*, **93,** 403.
Wilson, G. S. (1942). "The Pasteurization of Milk." Edward Arnold & Co., London.
——— (1932). Vet. Rec., **44,** 1226, 1240.

Brit. J. soc. Med. (1949), **3**, 56-68.

A STUDY ON BIRTHWEIGHT AND INTELLIGENCE

BY

CÉCILE ASHER* and J. A. FRASER ROBERTS

From the Institute of Child Health, University of London, and the Burden Mental Research Department, Stoke Park Colony, Bristol

INTRODUCTION

The mental development of the premature or immature child has been studied by a number of investigators. The literature has been summarized recently by one of us (Asher, 1946). But although there is a considerable body of information on the subsequent intelligence of children of low birthweight, so far as we are aware no study has been carried out on the general relation of birthweight to intelligence in the population as a whole. Doubtless this is due to the formidable difficulty of following up after a sufficient number of years groups of children whose weights were accurately measured at birth. But is there no other approach ? It is a matter of common experience that most mothers do in fact remember the birthweights of their children, often being able to quote them to the nearest ounce after the lapse of many years. The *a priori* objections to using this information are obvious enough. The mother of several children may get their birthweights mixed up. The weight as given to her may have been inaccurately determined. A proportion of mothers will have forgotten and the distribution of forgotten birthweights may not be the same as those that are remembered; there might well be a tendency, for example, for unusually high or low weights to be remembered more frequently than the rest. Nevertheless, it seems possible that for the comparison of the means of relatively large groups, weights as recollected by the mothers might be usable, for it is to be expected that errors of most types will be more or less randomly distributed. Accordingly it was decided to obtain from mothers the birthweights of a sample of children of school age. The distributions so obtained could then be compared with the distributions obtained from accurate hospital studies. Should this comparison prove satisfactory, the relation of birthweight to intelligence could then be examined, to a first approximation, by comparing the distributions of birthweights of children at schools and institutions of various types and containing children of very different mean intelligence.

It may be helpful to summarize here the results of our investigation. The frequency distributions of a sample of birthweights as recollected by the mothers correspond closely with those of an accurately measured hospital sample; there is no difference between schools of different type, containing children of very different mean intelligence; children at special schools for the educationally subnormal and certified mental defectives differ from the rest in showing a large excess of low birthweights and a small excess of very high birthweights, but there is no shifting of the frequency curve as a whole.

THE COMPOSITION OF THE SAMPLES

For the benefit of those who may not be familiar with the present English school system it should be explained that children first attend a primary school. Apart from the mentally defective and very backward, the primary schools contain children of all levels of intelligence. At the age of about 11+ the children proceed to a secondary school; the cleverest, as judged by a scholarship examination, or a group intelligence scale, or more usually both, pass on to a secondary grammar school, the remainder to a secondary modern school. From the point of view of intelligence the distinction is a sharp one. The mean Binet I.Q. of secondary grammar school children is about 120; that of secondary modern school children is about 95.

Children who are ascertained under the Education Act (1944) as being educationally subnormal and requiring special educational treatment may be admitted, at any stage of their school career, to special schools for the educationally subnormal. For brevity the term "special school" is used throughout this paper, and it will be understood that it is solely this kind of special school that is meant and not any of the other types provided for children handicapped in other ways. The proportion of children attending these special schools is

* Medical Officer, Ministry of Education

relatively small. Furthermore, the proportion differs considerably in different areas, for there is legitimate diversity of opinion as to the best way of catering for backward children. For example, in some areas many are accommodated in special classes at the ordinary schools. In the County of Middlesex the percentage of children (ages 5-16) at special schools is at present 0·38; in the London County Council area it is 0·73. The great majority of these children have Binet I.Q.s ranging from 50-75, rather less than 10 per cent. having I.Q.s outside this range. Selection is not made, however, solely on the basis of I.Q., though, of course, when a child has been submitted for examination on account of backwardness the I.Q. level is naturally an important factor in determining disposal.

The sample of children attending the ordinary schools was obtained from the Borough of Finchley, Middlesex. It should be mentioned that whereas the primary and secondary modern schools situated in the borough cater for Finchley children, there is a good deal of overlapping in regard to secondary grammar schools. In particular, the large boys' school shown in Table IV includes many pupils from outside the area; this explains the excess of boys in this portion of the sample.

There are four special schools in Middlesex and these formed the original special school sample. It was later decided to enlarge this important part of the material, so birthweights were obtained from seven additional special schools, situated in the London County Council area.

The sample of birthweights of certified mental defectives was obtained from Stoke Park Colony, Stapleton, Bristol.

Ascertainment of the Birthweights

Through the schools slips were distributed to the mothers of the children asking them to fill in the birthweight. Rather more than 80 per cent. of the slips were duly completed, the main reason for omission being, of course, that the mother had forgotten. The birthweights of the mental defectives were ascertained (with the help of the local health authorities) in the same way.

It was thought best simply to ask for the birthweight, without specifying how this should be stated: for example, in pounds and ounces, or to the nearest quarter-pound. This has naturally produced a rounding off of the figures, as is shown by the distribution of the first 5,177 birthweights (the whole sample, less the extra special schools and the mental defectives). The observed frequencies compared with those expected had measurements been made to the nearest ounce are shown in Table I.

TABLE I

Rounding-off of Birthweights (First 5,177) by Mothers

	Frequencies	
	As Given by Mother	Expected (Weights taken to nearest oz.)
To whole pound	1,375	323·6
To half-pound	1,399	323·6
To quarter-pound ..	583	323·6
To three-quarter pound ..	589	323·6
To intermediate ounces ..	1,231	3,882·7
Total	5,177	5,177·1

There was a strong tendency for weights to be given either to whole pounds or half-pounds, but fortunately none for whole pounds to be given more often than half-pounds; there was a slighter tendency to round off to the quarters and three-quarters. Thus the material can be grouped in one way only: namely, in half-pound groups with whole and half-pounds at the group centres. The quarter and three-quarter observations were distributed between the groups above and below in proportion to the numbers in those adjacent groups. The figures used for doing this were obtained from the whole original sample of 5,177; girls and boys were kept separate. The same figures were used for distributing the quarters and three-quarters amongst the additional special school sample and the mental defectives. The frequency distributions obtained are shown in Table II. The fractional frequencies are, of course, due to the distribution of the quarters and three-quarters.

Comparison of the Birthweights with an Accurately Measured Sample

An excellent sample of birthweights with which to compare the present material is that of Martin (1931). His series was composed of live births at Queen Charlotte's Hospital, London, during the years 1922-25. Thus both samples consist of children drawn from Greater London; the numbers are fairly similar; twins are included in both. The one important difference is that Martin's sample included all live births, while ours represents survivors to various school ages. In our sample the special school and mentally defective children are heavily over-represented (this fraction of the child

TABLE II

Frequency Distribution of Birthweights*

Group Central Weights (lb.)	Ordinary Schools				Special Schools	Mental Defectives		
	Primary	Secondary Grammar	Secondary Modern	Total		Feeble-Minded	Imbeciles	Idiots
				Girls				
1·5	—	—	—	—	—	—	1·0	—
2·0	1·0	—	—	1·0	2·0	—	—	—
2·5	1·0	—	7·7	8·7	3·0	1·0	—	—
3·0	2·0	—	0·5	2·5	6·0	—	2·0	—
3·5	5·4	—	10·2	15·6	6·8	—	1·4	1·0
4·0	11·4	2·8	12·4	26·6	6·4	—	2·6	1·0
4·5	21·7	6·6	10·3	38·6	10·8	1·0	1·7	—
5·0	38·1	11·0	13·8	62·9	18·2	—	4·3	3·4
5·5	46·2	9·5	22·7	78·4	19·3	—	3·3	2·9
6·0	118·6	23·5	48·5	190·6	39·3	—	2·7	3·6
6·5	162·8	46·4	79·5	288·7	46·6	1·0	2·8	5·1
7·0	206·5	65·5	109·1	381·1	58·0	3·0	1·2	7·5
7·5	200·4	76·0	118·6	395·0	57·9	2·0	4·2	5·1
8·0	165·3	50·1	86·6	302·0	30·5	2·0	5·4	5·4
8·5	128·4	36·2	68·1	232·7	30·3	1·0	3·4	2·0
9·0	40·5	16·4	27·6	84·5	17·9	2·0	1·0	2·0
9·5	24·9	12·0	15·8	52·7	10·0	1·0	2·0	—
10·0	19·0	5·0	16·2	40·2	5·4	1·0	1·0	—
10·5	3·8	3·0	0·4	7·2	1·6	—	—	—
11·0	1·0	—	1·5	2·5	3·0	—	—	0·5
11·5	1·0	—	1·5	2·5	—	—	—	0·5
12·0	1·0	—	2·0	3·0	1·0	—	—	—
12·5	—	—	1·0	1·0	1·0	—	—	—
Total	1200·0	364·0	654·0	2218·0	375·0	15·0	40·0	40·0

Group Central Weights (lb.)	Ordinary Schools				Special Schools	Mental Defectives		
	Primary	Secondary Grammar	Secondary Modern	Total		Feeble-Minded	Imbeciles	Idiots
				Boys				
1·5	—	—	—	—	1·0	—	—	—
2·0	—	—	—	—	1·0	—	—	—
2·5	1·0	—	—	1·0	1·0	—	3·0	0·4
3·0	2·0	1·0	1·0	4·0	1·0	1·0	—	0·6
3·5	1·0	4·0	1·0	6·0	6·5	—	—	—
4·0	9·4	0·3	1·3	11·0	4·5	—	2·0	2·0
4·5	10·8	3·7	5·7	20·2	8·9	—	3·0	1·3
5·0	23·6	18·3	14·4	56·3	18·5	4·0	2·9	4·2
5·5	31·8	20·2	14·7	66·7	17·3	—	6·1	4·8
6·0	78·6	32·5	37·0	148·1	37·4	2·4	10·2	5·4
6·5	134·5	70·8	70·6	275·9	56·7	2·0	21·4	11·5
7·0	195·0	86·7	103·0	384·7	80·0	5·0	12·4	12·7
7·5	221·9	138·7	134·6	495·2	64·3	2·6	15·9	7·2
8·0	160·0	102·6	106·8	369·4	65·4	4·0	21·7	12·4
8·5	145·5	70·2	77·0	292·7	54·1	3·0	11·4	5·6
9·0	91·3	54·5	73·3	219·1	28·1	3·0	10·3	5·9
9·5	43·2	27·7	29·1	100·0	22·8	—	4·2	2·0
10·0	34·8	23·4	25·9	84·1	21·5	1·0	7·5	—
10·5	16·4	4·4	12·0	32·8	5·8	—	5·0	0·8
11·0	5·9	1·0	1·6	8·5	3·2	1·0	—	0·9
11·5	0·3	1·6	2·0	3·9	—	1·0	—	0·9
12·0	—	0·4	1·0	1·4	2·0	—	1·0	0·4
12·5	—	—	—	—	1·0	—	—	—
13·0	—	—	1·0	1·0	—	1·0	—	—
Total	1207·0	662·0	713·0	2582·0	502·0	31·0	138·0	79·0

* Fractional frequencies are due to distribution between adjacent groups of weights given to ¼ lb. and ¾ lb.

population is numerically very small) so they have been omitted altogether in making the present comparison. Martin shows separately first pregnancies, second pregnancies, and subsequent pregnancies. Order of birth was not ascertained in our sample, so it is necessary to weight his figures, as given in his Table I, in order to arrive at single means and standard deviations. In a survey conducted at Bath (Roberts, Norman, and Griffiths, 1938) the birth order of 3,300 schoolchildren was ascertained. Making some allowance for previous stillbirths and deaths, the following weights seem reasonable: first children, 0·33; second, 0·27; third and subsequent, 0·40. Even if these weights are somewhat in error for the present sample of children, the differences in the weighted total figures for Martin's series will be very small. Anticipating the next section of this paper, no appreciable difference was found between children attending the various types of school (other than special schools); hence these can be added together. The comparison is shown in Table III.

TABLE III

COMPARISON OF BIRTHWEIGHTS OF NORMAL SAMPLE WITH MARTIN'S ACCURATE SAMPLE

	Mean	Standard Deviation
	Girls	
Martin	7·14	1·19
Present sample	7·21	1·30
	Boys	
Martin	7·47	1·24
Present sample	7·60	1·27

The omission of the special school children and the mental defectives from our sample could not, as the proportions of such children are so small, make a difference of as much as one in the last figures shown. A much more important matter is that Martin's sample includes all live births, ours only survivors to various school ages; this must account for some of the difference in means. Allowing for this factor, there is close similarity. In regard to dispersion, the boys of the two series are very similar, but our girls are somewhat more variable; this is due to four of the schools showing an over-variable frequency distribution of birthweights of girls, as is discussed in the following section.

The frequency distributions for the girls and boys at ordinary schools, as shown in Table I above, show considerable departures from the normal form. In terms of 3rd and 4th moments, the girls show:

$g_1 = -0{\cdot}3131 \pm 0{\cdot}0520$; $g_2 = +1{\cdot}1952 \pm 0{\cdot}1039$

and the boys:

$g_1 = -0{\cdot}0765 \pm 0{\cdot}0482$; $g_2 = +0{\cdot}6252 \pm 0{\cdot}0963$

Thus in both sexes there is negative skewness, highly significant in the girls, but not attaining the 5 per cent. level in the boys. There is marked and highly significant positive kurtosis in both sexes.

Martin's frequency distributions are not given in a form permitting similar calculations, but it is possible from his Table IV to compare observed and expected numbers falling into broad groups. It should be mentioned that his Table IV comprises 3,443 boys and 3,269 girls, whereas the means and standard deviations have to be based on the somewhat larger numbers of his Table I, 3,526 boys and 3,329 girls. This cannot, however, make any appreciable difference. In assembling our birthweights into similar broad groups, the group boundaries cannot be made the same as his: $4\frac{3}{32}\frac{1}{2}$, $5\frac{3}{32}\frac{1}{2}$ . . . $9\frac{3}{32}\frac{1}{2}$. Ours have to be the slightly higher figures: $5\frac{1}{4}$, $6\frac{1}{4}$ . . . $10\frac{1}{4}$. In view of our higher means, however, this is probably not a disadvantage. The comparison is shown in Table IV.

The closeness of the correspondence is remarkable, especially in the girls. It is clear that the frequency distribution of human birthweights departs considerably from the normal form, being negatively skewed with positive kurtosis; our series has faithfully reflected this fact.

To sum up the results of this section, a sample of birthweights based on the recollections of mothers of children of various school ages, with all the omissions due to lack of knowledge or forgetfulness, and including all the errors that might have been anticipated, has yielded a frequency distribution which corresponds closely with that of a sample of highly accurate birthweights obtained at a maternity hospital: in means and dispersions showing reasonable agreement; in the actual form of the curve, in type and amount of departure from normality, a remarkably close correspondence. There seems every reason to anticipate that birthweights obtained in this simple and trouble-free way should be reliable enough for the comparison of relatively large groups. So, heartened by this result, we can now proceed with some confidence to examine differences between groups within our sample.

ANALYSIS OF THE DIFFERENCES BETWEEN SCHOOLS (OTHER THAN SPECIAL SCHOOLS)

In addition to examining the differences between the three types of school containing normal children it seemed advisable to examine also the differences

TABLE IV

DEPARTURES FROM NORMALITY: MARTIN'S SERIES AND PRESENT SERIES

Martin's Series					Present Series				
Group boundaries (lb.)	Exptd. no.	Obs. no.	Obs.-exptd.	χ^2	Group boundaries (lb.)	Exptd. no.	Obs. no.	Obs.-exptd.	χ^2
				Girls					
$4\frac{31}{32}$	111·4	125	+13·6	1·660	$5\frac{1}{4}$	144·8	155·9	+11·1	0·851
$5\frac{31}{32}$	446·1	361	−85·1	16·234	$6\frac{1}{4}$	363·2	269·0	−94·2	24·432
$6\frac{31}{32}$	974·1	910	−64·1	4·218	$7\frac{1}{4}$	625·8	669·8	+44·0	3·094
$7\frac{31}{32}$	1039·1	1,191	+151·9	22·205	$8\frac{1}{4}$	613·0	697·0	+84·0	11·511
$8\frac{31}{32}$	541·8	543	+1·2	0·003	$9\frac{1}{4}$	341·6	317·2	−24·4	1·743
$9\frac{31}{32}$	138·3	122	−16·3	1·921	$10\frac{1}{4}$	108·1	92·9	−15·2	2·137
	18·2	17	−1·2	0·079		21·5	16·2	−5·3	1·307
	3269·0	3,269		46·320		2218·0	2218·0		45·075
				Boys					
$4\frac{31}{32}$	77·8	78	+0·2	0·001	$5\frac{1}{4}$	81·9	98·5	+16·6	3·365
$5\frac{31}{32}$	345·1	305	−40·1	4·660	$6\frac{1}{4}$	287·3	214·8	−72·5	18·295
$6\frac{31}{32}$	866·4	810	−56·4	3·671	$7\frac{1}{4}$	640·1	660·6	+20·5	0·657
$7\frac{31}{32}$	1117·9	1,195	+77·1	5·317	$8\frac{1}{4}$	786·9	864·6	+77·7	7·672
$8\frac{31}{32}$	738·2	760	+21·8	0·644	$9\frac{1}{4}$	536·7	511·8	−24·9	1·155
$9\frac{31}{32}$	250·3	242	−8·3	0·275	$10\frac{1}{4}$	201·9	184·1	−17·8	1·569
	47·3	53	+5·7	0·687		47·2	47·6	+0·4	0·003
	3443·0	3,443		15·255		2582·0	2582·0		32·716

Degrees of freedom=4; Martin, boys, $P<0{\cdot}01$; remainder, $P<0{\cdot}001$.

between schools of the same type, thus making some assessment of the internal consistency of the figures. The means and standard deviations for the 14 schools are shown in Table V. Thirteen were mixed schools; one was a boys' school.

An analysis of the variation of the means is presented in Table VI.

It will be seen that the school means for the girls do not reveal any evidence of significant heterogeneity, either between school types, or between schools within school types. There is significant, though not highly significant, variation between school types amongst the boys. This is due to the secondary modern boys (i.e. the dullest) being somewhat heavier than the remainder. As, however, the secondary modern girls attending the same schools are slightly lighter than the remainder, it does not seem necessary to attach any importance to this moderate discrepancy. As in the girls, schools of the same type are homogeneous.

The homogeneity of the variances may be examined by Bartlett's method (1937). For the boys $-2\log\mu$ is 5·22; this is distributed as χ^2 for 13 degrees of freedom and so the school variances are perfectly homogeneous. It is the more remarkable to find, therefore, that the school variances of the girls are distinctly heterogeneous; $-2\log\mu$ is 42·33, which with 12 degrees of freedom is highly significant. Actually, the heterogeneity is entirely due to four schools, one primary and three secondary modern, which are far more variable in their birthweights than the rest. This, of course, makes the secondary modern birthweights significantly more variable than those of the other types of school

TABLE V

BIRTHWEIGHTS OF NORMAL CHILDREN. NUMBERS, MEANS, AND STANDARD DEVIATIONS, SEPARATELY BY SCHOOLS

	Girls			Boys		
School	No.	Mean	Standard Deviation	No.	Mean	Standard Deviation
Primary						
1	245	7·270	1·149	221	7·429	1·260
2	113	7·151	1·218	135	7·559	1·305
3	236	7·133	1·325	254	7·638	1·258
4	180	7·119	1·199	184	7·510	1·228
5	99	7·209	1·237	117	7·624	1·363
6	173	7·116	1·212	151	7·523	1·248
7	154	7·245	1·387	145	7·527	1·245
Total	1,200	7·179	1·245	1,207	7·542	1·266
Secondary Grammar						
1	184	7·367	1·162	153	7·615	1·196
2	180	7·307	1·184	166	7·687	1·310
3	—	—	—	343	7·534	1·250
Total	364	7·337	1·172	662	7·591	1·253
Secondary Modern						
1	203	7·239	1·472	193	7·786	1·263
2	122	7·393	1·283	168	7·716	1·283
3	212	7·072	1·531	236	7·702	1·318
4	117	7·206	1·423	116	7·591	1·199
Total	654	7·208	1·451	713	7·710	1·275
Total—all schools	2,218	7·213	1·299	2,582	7·601	1·267

TABLE VI

ANALYSIS OF VARIANCE. CHILDREN ATTENDING NORMAL SCHOOLS

Girls

Variation of Birthweight $(lb.)^2$	Degrees of Freedom	Sum of Squares	Mean Square	Variance Ratio	P
Between school types	2	7·048	3·524	2·09	>0·05
Between schools within school types ..	10	13·309	1·331	—	—
Within schools	2,205	3719·856	1·687	—	—
Total	2,217	3740·213			
Between schools, all types	12	20·357	1·696	1·01	—

Boys

Variation of Birthweight $(lb.)^2$	Degrees of Freedom	Sum of Squares	Mean Square	Variance Ratio	P
Between school types	2	12·693	6·347	3·96	0·05—0·01
Between schools within school types ..	11	11·807	1·073	—	—
Within schools	2,568	4115·577	1·603	—	—
Total	2,581	4140·077			
Between schools, all types	13	24·500	1·885	1·18	—

and also the whole sample of girls more variable than the boys. If the three largest variances are omitted $-2\log\mu$ is reduced to 12·10 and so becomes non-significant. It is very curious that the girls at these four schools should be so variable in birthweight, whereas the boys attending the same four schools—drawn from the same areas and often from the same families—should show no such difference. We have no explanation to offer and the finding remains a disturbing one.

Reviewing the evidence as a whole we are disposed to conclude that it is safe to use the material for the comparison of large groups; the homogeneity of the school means in the girls, with only a slight departure in regard to school types in the boys, and the homogeneity of the school variances in the boys, do seem to point to a body of data that is sufficiently reliable for this purpose, even though four schools are over-variable in the birthweights of the girls. And, of course, there is the more important fact of close correspondence with Martin's accurate measurements dealt with in the preceding section.

Lack of Association between Birthweight and Intelligence in Normal Schoolchildren

The results of the preceding section show that there is no appreciable difference between the mean birthweights of children attending the three types of school. To summarize the figures:

Type of School	Mean Birthweight (lb.)	
	Girls	Boys
Primary	7·18	7·54
Secondary Grammar ..	7·34	7·59
Secondary Modern ..	7·21	7·71

If there were an association between high intelligence and high birthweight, primary school children would be expected to give a mean figure representative of the whole population, whereas secondary grammar school children would be higher and secondary modern children lower. Yet with a difference in mean intelligence of well over a standard deviation there is no indication of any corresponding difference in birthweight. It can be safely concluded, therefore, that in the general normal population there can be no appreciable correlation between birthweight and intelligence.

Anticipating the next section, it may be asked whether there is any indication that there is a higher proportion of low birthweights amongst the secondary modern children compared with the secondary grammar school children. The figures show:

	Percentage of children below 6·25 lb. (boys) and 5·75 lb. (girls)
Primary	11·8
Secondary Grammar	10·7
Secondary Modern ..	11·2

	Percentage of children below 5·25 lb. (boys) and 4·75 lb. (girls)
Primary	3·8
Secondary Grammar	3·6
Secondary Modern ..	4·7

This last difference between the secondary grammar and secondary modern schools is not significant ($\chi^2=1·89$). Some caution is needed in interpreting this finding. The proportion of children at special schools is so small that even if they were all included with the secondary modern children, the figures

TABLE VII

Birthweights of Special School Children. Numbers, Means, and Standard Deviations, separately by Schools

School	Girls			Boys		
	No.	Mean	Standard Deviation	No.	Mean	Standard Deviation
1	52	6·605	1·401	74	7·105	1·578
2	41	6·362	1·638	64	7·509	1·121
3	33	7·206	1·785	62	7·258	1·617
4	23	7·630	1·382	28	7·861	1·764
5	25	6·942	1·564	32	7·447	1·536
6	27	6·922	1·455	49	6·920	1·734
7	60	7·133	1·762	—	—	—
8	51	6·878	1·628	—	—	—
9	63	6·869	1·712	—	—	—
10	—	—	—	128	7·459	1·625
11	—	—	—	65	7·756	1·344
Total	375	6·905	1·633	502	7·396	1·552

would not be appreciably changed. Secondary modern schools contain some children whose intelligence is almost as low, or fully as low, as that of some children at special schools. It cannot be deduced that this small fraction of the very dull does not include an undue proportion of very low birthweights; relatively enormous numbers would be needed, however, to decide the point. What can be deduced is that the separation of children into the brighter fraction destined for secondary grammar schools and the duller fraction destined for secondary modern schools has no counterpart in any appreciable excess of low birthweights in the latter type of school. In other words, a measurable excess of low birthweights is found only in that portion of the range comprising relatively very low intelligences.

Homogeneity of the Special Schools in Regard to Birthweight

Eleven special schools have been included in this study, three being girls' schools, two boys' schools, and six mixed schools. The means and standard deviations are shown separately by schools in Table VII.

Table VIII shows the analysis of variance.

Again using Bartlett's method for testing the homogeneity of the variances, $-2 \log\mu$ is 5·51 for the girls and 16·09 for the boys, distributed as χ^2 for 8 and 7 degrees of freedom respectively.

The girls are therefore perfectly homogeneous in regard both to school means and variances. With the boys there is heterogeneity which just attains the 0·05 level of significance for school means and somewhat exceeds it for variances. This seems a satisfactory result, as some evidence of heterogeneity, considerably more pronounced than has actually been found, would not have been surprising. The special schools cover a wide area and in view of variations in local policy it might well be that different schools tend to receive rather different types of pupil.

Birthweights of Special School and Mentally Defective Children

In view of the uniformity of the mean birthweights of children at primary, secondary grammar, and secondary modern schools, these can be added together for comparison with the birthweights of the special school children and the certified mental defectives. The comparison is shown in Table IX. The retarded children are lighter and more variable. The differences between the normal children and the special school samples are significant, highly so in the boys. The numbers of mental defectives are not sufficient for the differences to attain the level of significance, but the same trend is plain.

Inspection of Table II shows that these differences are not due to a shifting of the whole curve, but to a large excess of very low birthweights amongst the backward children, together with a much smaller excess of very large weights. Within the region 6·25-10·75 lb. in the boys and 5·75-10·25 lb. in the girls all three distributions are practically identical. This comparison is shown in Table X.

If, then, the low and very high weights are omitted,

TABLE VIII

Analysis of Variance (Children attending Special Schools)

Girls

Variation of Birthweight (lb.)²	Degrees of Freedom	Sum of Squares	Mean Square	Variance Ratio	P
Between schools	8	35·126	4·391	1·67	>0·05
Within schools	366	962·548	2·630		
Total	374	997·674			

Boys

Variation of Birthweight (lb.)²	Degrees of Freedom	Sum of Squares	Mean Square	Variance Ratio	P
Between schools	7	34·436	4·919	2·07	0·05
Within schools	494	1172·686	2·374		
Total	501	1207·122			

TABLE IX

BIRTHWEIGHTS OF NORMAL, SPECIAL SCHOOL, AND MENTALLY DEFECTIVE CHILDREN. NUMBERS, MEANS, AND STANDARD DEVIATIONS

	Girls			Boys		
Type of School	No.	Mean	Standard Deviation	No.	Mean	Standard Deviation
Normal ..	2,218	7·213	1·299	2,582	7·601	1·267
Special School	375	6·905	1·633	502	7·396	1·552
Mentally Defective	95	6·752	1·826	248	7·368	1·698

TABLE X

COMPARISON WITHIN BIRTHWEIGHT RANGES 6·25-10·75 LB. (BOYS) AND 5·75-10·25 LB. (GIRLS) OF NORMALS, SPECIAL SCHOOL PUPILS, AND MENTAL DEFECTIVES

	Girls			Boys		
Type of School	No.*	Mean	Standard Deviation	No.*	Mean	Standard Deviation
Normal ..	1967·5	7·475	0·949	2253·9	7·876	0·978
Special School	295·9	7·403	1·006	398·7	7·874	1·043
Mentally Defective ..	67·4	7·605	1·031	188·5	7·866	1·083

* Fractional frequencies are due to distribution between adjacent groups of weights given to ¼ lb. and ¾ lb.

the differences in means vanish, none being significant. The backward children are still slightly more variable, but the differences are very small compared with those shown for the total frequency distributions in Table IX. Adding together the special school and mentally defective children, the variances are as follows:

	Girls	Boys
Normal children ..	0·9011	0·9557
Backward children	1·0255	1·1132
Difference	0·1244 ±0·0732	0·1575 ±0·0632

The difference is not significant in the girls, but in the boys it is 2·49 times its standard error. The curtailing of the frequency distributions has, of course, produced marked negative kurtosis, so reducing the standard errors of the variances. This has been allowed for by using the fourth cumulants.

Thus over a portion of the range that includes some 88 per cent. of normal children, there is no difference in means between normal children, special school children, and certified mental defectives. The backward children are still very slightly more variable, but the omission of the low birthweights and of a very small proportion of high birthweights has reduced the original large differences to very small limits.

Turning now to the birthweights below 6·25 lb. in boys and 5·75 lb. in girls, we find the following percentages below these limits:

	Girls	Boys	Both Sexes
Normal ..	10·6	12·1	11·4
Special School ..	19·3	19·3	19·3
Mental Defectives	30·7	21·2	23·7

The excess of low birthweights amongst both kinds of backward children is very pronounced. (It should be noted that the number of mentally defective girls is very small.)

In order to make a finer comparison it seems best to treat both sexes together, adding the small sample of feeble-minded to the special school sample (the range of I.Q.s is not very dissimilar) and also taking idiots and imbeciles together, as they are too few to separate usefully. The results are shown in Table XI. The same figures are shown in Table XII, but this time as relative proportions, the percentage of birthweights of normal children falling into each frequency group being taken as 100.

There is a steady increase in the disproportion as

TABLE XI

COMPARISON OF PERCENTAGES OF LOW BIRTHWEIGHTS IN NORMAL AND RETARDED CHILDREN

Group Boundaries (lb.)		Normal	Special School and Feeble-minded	Idiots and Imbeciles
Girls	Boys	(Total 4,800)	(Total 923)	(Total 297)
4·75—5·75	5·25—6·25	7·42	10·25	13·60
3·75—4·75	4·25—5·25	2·95	5·37	5·62
2·75—3·75	3·25—4·25	0·73	2·58	2·83
<2·75	<3·25	0·31	1·19	1·68
<5·75	<6·25	11·41	19·39	23·73

TABLE XII

PROPORTIONS OF LOW BIRTHWEIGHTS IN RETARDED CHILDREN WITH PROPORTIONS IN NORMALS FIXED AT 100

Group Boundaries (lb.)		Normal	Special School and Feeble-minded	Idiots and Imbeciles
Girls	Boys			
4·75—5·75	5·25—6·25	100	138	183
3·75—4·75	4·25—5·25	100	182	191
2·75—3·75	3·25—4·25	100	353	388
<2·75	<3·25	100	384	542
<5·75	<6·25	100	170	208

birthweight falls. There are four times as many birthweights under 2·75 lb. for girls and 3·25 lb. for boys as in the normal series and this disproportion becomes steadily less with each increase of one pound in weight.

The difference between special school children and the feeble-minded on the one hand and idiots and imbeciles on the other is smaller than might have been anticipated. There does appear, however, to be a slightly higher proportion of small birthweights amongst the low grade defectives, but our series is too small to pursue this point further. The chief finding, as far as our figures go, is that an excess of low birthweights seems to be almost as strongly associated with high grade mental deficiency as with idiocy and imbecility.

Examination of Table I suggests that perhaps one should go a little higher than the limits chosen in this paper before all discrepancy vanishes; but to add another half-pound, including as low weights girls under 6·25 lb. and boys under 6·75 lb., would make the comparison worse, not better, as is shown by the first results of this section. Similarly, half a pound is slightly too big a difference to make between the sexes. In these matters, however, we are limited by our grouping of birthweights, to which there was no alternative.

It remains to compare the very high weights, which we have fixed, after inspection of the frequency distributions, as above 10·25 lb. for girls and 10·75 lb. for boys. Here the figures are so small that we have added together the special school and mental defective samples. The comparison is shown in Table XIII.

It seems best to compare the large birthweights with those within that range over which normal and backward children do not differ. The excess of large weights amongst the backward children is significant in the girls ($\chi^2=4·74$) and highly so in the boys ($\chi^2=10·12$). It should be recalled, however, that the mothers of the backward children are themselves, on the average, of lower intelligence than the mothers of the normal series, and so there might be rather more mistakes amongst the birthweights as given by them. It is very unlikely indeed that the difference can be explained in this way, but with small absolute numbers it is perhaps necessary to be cautious and to await the collection of more ample data before accepting this finding without some reservation.

TABLE XIII

COMPARISON OF NORMAL AND BACKWARD CHILDREN OF VERY LARGE WEIGHTS

	Girls			Boys		
	No.* 5·75—10·25 lb.	No.* over 10·25 lb.	Percentage over 10·25 lb.	No.* 6·25—10·75 lb.	No.* over 10·75 lb.	Percentage over 10·75 lb.
Normal ..	1967·5	16·2	0·82	2253·9	14·8	0·65
Backward ..	363·3	7·6	2·05	587·2	12·4	2·07

* Fractional frequencies are due to distribution between adjacent groups of weights given to ½ lb. and ¾ lb.

LOW AND HIGH BIRTHWEIGHTS IN RELATION TO MENTAL RETARDATION

It would seem, then, if our conclusions are soundly based, that there is no appreciable general relation between birthweight and intelligence; even idiots and imbeciles show no tendency to be lighter at birth than gifted children; there is no shifting of the frequency curve. But mental defectives and very backward children do include a substantial excess of low birthweights and a small excess of very high ones. It is tempting, though not of course justifiable on the data of this paper, to translate these findings into the simple statement that very backward children differ from others in regard to birthweight simply in being more often premature or postmature.

Considering the low weights first, this study provides no evidence as to whether babies destined to be mentally defective tend to be more often premature than do normal children, or whether the prematurity (or immaturity) is the cause of the mental deficiency. In all probability both are true. Prematurity with its associated fragile blood-vessels predisposes to intracranial haemorrhage, and it would not be surprising to find that the bulk of the excess is due to mental deficiency which is a consequence of the prematurity. If the whole of the excess were to be explained in this way, our figures would indicate that some 7 per cent. of mental deficiency amongst children of school age is to be ascribed to this cause. This is likely to prove an overestimate; but we do feel that a smaller, though not negligible, fraction may well be so caused. If this is true, it is an important fraction, for unlike so much else in the causation of mental deficiency, this is a factor which offers the promise of control and improvement.

As regards the excess of very large babies amongst the defectives, it is difficult not to ascribe this directly to the resulting difficult labour and greater liability to birth injury. Our figures indicate that 1 per cent. of mental deficiency may be caused in this way.

It has already been pointed out that the defective and special school fractions are relatively small numerically. Hence the observed excess of low weights amongst these children does not mean a bad prognosis as regards subsequent mentality for the individual premature child. Even below 3·25 lb. for girls and 2·75 lb. for boys, where the percentage is multiplied fourfold as compared with normal schoolchildren, the likelihood that any given child below these limits will be mentally defective should not exceed 10 per cent. But it is not without significance, perhaps, that of the only two children with birthweights of 1½ lb. in our 6,000 (both checked and found to be correct), one was at a special school and the other was an imbecile.

These findings seem to agree well with studies on the development of immature or premature babies. As already mentioned, the literature has recently been summarized by one of us (Asher, 1946). On the whole there is a rather remarkable agreement on the facts, namely, that a proportion of such children which may be estimated very roughly at 10 per cent. is mentally defective or very backward. To select one careful study as an example, Rosanoff and Inman-Kane (1934) found that 10·3 per cent. of their 381 subjects had I.Q.s below 76. Where the various writers differ is not so much on the facts, but in the point of view from which they regard them. Some, the pessimists, point out that the proportion of mental deficiency is far higher than in children of normal birthweight; others, the optimists, are more impressed by the fact that any given premature child is much more likely to grow up with normal mentality than to be mentally defective.

A striking, and perhaps surprising, point about the present study is that low birthweight seems to be almost if not quite as strongly associated with feeble-mindedness and pronounced backwardness

as it is with gross damage resulting in idiocy or imbecility. But once again this is in harmony with the findings in the previous study (Asher, 1946) and apparently with the literature generally.

It may not be out of place to say a word about the accepted figure of 5½ lb. as marking the limit of immaturity. First, this study, as well as others, indicates that a sex difference should be made. Karn (1947) suggests that the limit should be 0·25 lb. higher for boys; the present study indicates a slightly larger difference, about 0·4 lb. Further refinements might well include adjustments for parity and also in the case of twins, though these are somewhat less important. Secondly, at least from the point of view of some association with subsequent mental impairment, the accepted limit is a little low. As has been shown above, there is evidence of some unfavourable effect up to about 5·75 lb. for girls and about 6·25 lb. for boys.

As regards low and high weights and their association with mental deficiency, this is a preliminary study. We have made no attempt to subdivide the defective children by types, or, except very roughly, by grade. Our figures are too small for this. Far larger numbers are needed and, coupled with this, much more detail. There would seem to be good indications that further studies on these lines should lead to useful results.

Summary

1. A sample of 4,800 birthweights of children attending primary, secondary grammar, and secondary modern schools was obtained by questioning the mothers. The frequency distributions corresponded very closely to those of Martin's series of birthweights accurately measured at a maternity hospital; the resemblance between the two series extended to means, dispersions, and to the type and amount of departure from normality. The individual school means and variances were satisfactorily homogeneous except that for some unexplained reason the girls at four schools were over-variable. It is concluded that birthweights obtained in this way can safely be used for certain purposes, such as the comparison of the means of large groups.

2. There was no appreciable difference between the birthweights of children attending the three types of school. As secondary grammar and secondary modern children differ by more than a standard deviation of Binet I.Q., it is concluded that there can be no appreciable association between birthweight and intelligence in the general population taken as a whole.

3. A sample of birthweights of children attending special schools for the educationally subnormal, and also a sample of certified mental defectives, had lower mean birthweights and also showed more variability than the normal children. These differences are entirely due to an excess of very low birthweights amongst the backward children, together with a very much smaller excess of very large birthweights. Between the limits 5·75 lb. to 10·25 lb. for girls and 6·25 lb. to 10·75 lb. for boys (a range which includes 88 per cent. of birthweights of normal children) there was no difference between the normal and the backward series.

4. The excess of low birthweights amongst the backward children increases steadily as birthweight falls. The excess is not more than about 50 per cent. in the region 4·75 lb. to 5·75 lb. for girls and 5·25 lb. to 6·25 lb. for boys. But there are about four times as many birthweights below 2·75 lb. and 3·25 lb. respectively in the backward children as there are amongst the normals. The excess of low birthweights is little greater amongst idiots and imbeciles than it is amongst the feeble-minded and borderline defectives.

5. The fact that special school children and certified defectives form a relatively small proportion of the population means that the figures just quoted indicate that the child of low birthweight is much more likely to be of normal mentality than to be defective or very backward. The chance for those of the lowest birthweights, as quoted above, is probably not worse than 10 per cent.

We are greatly indebted to the Middlesex County Council and the London County Council for the facilities made available at the schools, and in particular we should like to acknowledge the generous assistance of Dr. A. A. Turner, School Medical Officer of the Borough of Finchley, and all the head teachers at the Finchley schools and at the additional special schools for educationally subnormal children situated in the wider areas. The birthweights of the sample of certified mental defectives were very kindly provided by Dr. R. M. Norman, Medical Superintendant of Stoke Park Colony, Bristol, who had obtained them during the course of a much more detailed investigation into the birth histories of the younger patients under his care. We are indebted to Mrs. M. G. Young for preparing the figure.

References

Asher, C. (1946). *Brit. med. J.*, **1**, 793.
Bartlett, M. S. (1937). *Proc. roy. Soc.*, **A160**, 268.
Karn, M. N. (1947). *Ann. Eug., Camb.*, **14**, 44.
Martin, W. J. (1931). *Ibid.*, **4**, 327.
Roberts, J. A. F., Norman, R. M., and Griffiths, R. (1938). *Ibid.*, **8**, 178.
Rosanoff, A. J., and Inman-Kane, C. V. (1934). *Am. J. of Psychiat.*, **13**, 829.

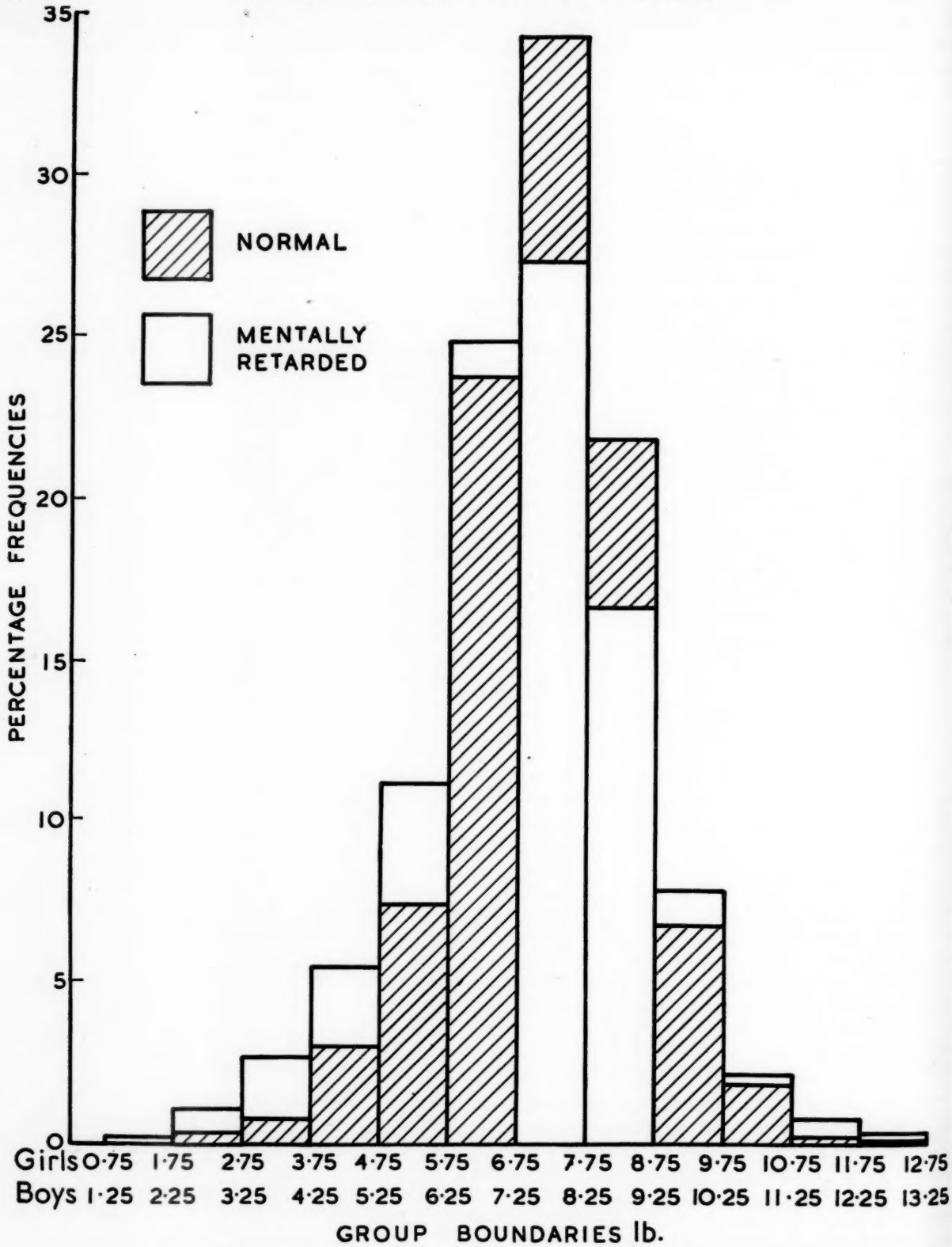

FIG. 1.—Comparison of percentage frequency distributions of birthweights of 4,800 normal schoolchildren and 1,220 mentally retarded children (special school and certified defectives).

Brit. J. soc. Med. (1949), **3**, 69-76

CONTROL CHARTS FOR THE STANDARD MORTALITY RATIO

BY

G. HERDAN

From the Department of Preventive Medicine, University of Bristol

The Registrar General for England and Wales tells us not only how fast our species is killed, but what kills it, and the preference a specified disease has for different age-groups and for different occupations. The selectivity in the action of the various killers of our species with regard to social status and occupation has found expression in the so-called Standard Mortality Ratio (S.M.R.) which is the index the Registrar General uses for the purpose of describing the selective action of diseases.

The S.M.R. may be briefly explained thus. The Registrar General calculates " standard deaths," which are the numbers obtained by applying the general mortality rates of Table 3 of the *Registrar General's Decennial Supplement England and Wales, Part IIA, Occupational Mortality*, 1931, for all males, all married women, or all single women, as the case may be, at the appropriate age-groups, 20-25-35-45-55-65, to three times the census population of the occupational group as given in Table 4 of the Report, and summing the products. They represent the deaths which would result in an occupation group if that group were exposed at each age to the standard mortality risks. The S.M.R. is the percentage ratio of the deaths actually registered for the group to the calculated standard deaths.

There is, however, apart from systematic and accidental errors of diagnosis, a type of error to be taken into account in comparing S.M.R.s. This is the error due to the fact that the people following a certain occupation form only a comparatively small sample of the total population. Such a sample, if taken at random, may include a greater or smaller number of persons sensitive to the disease in question, or to disease in general. That number, provided the sample is truly random, is due to chance only. Any increase in the S.M.R. due only to the inclusion of a greater number of sensitive persons must not be put down as due to the occupation or social status. We must, therefore, allow for chance fluctuations in the S.M.R. of a population group before judging its S.M.R. for significance.

There will be a certain probability for a deviation of a specified magnitude to occur due to chance only. The deviation of the S.M.R. from 100 per cent. must be considered in the light of these chance deviations (Yule, 1934). The Registrar General's method of doing this is described in his *Decennial Supplement; Part IIA*. A table is given from which to read the limits within which a difference in the S.M.R. cannot be considered significant or highly significant. These limits vary with the number of deaths in the population group, and thus with its size. In his discussion of the various S.M.R.s the Registrar General takes the standard error into account. But since the report of the Registrar General comprises 176 great folio pages of text and 228 similar pages of tables, some measure of condensation would appear desirable for doctors, and particularly for medical officers, who either cannot spare the time for study, or have not the mathematical stamina for wading through large numerical tables and at the same time applying the criterion of the multiples of the standard errors to every S.M.R. in which they happen to be interested. It is with this in mind that the following charts have been constructed. The quality control chart (Herdan, 1948) is here introduced as a device for facilitating the use of the standard error in judging the significance of S.M.R.s.

The general structure of these charts is as follows.

A straight line represents the S.M.R. for the total population (all males, all married women, all single women, as the case may be) for all causes or for specified diseases. The curved lines are drawn at distances representing 1·5 and 2·5 of the standard error of the S.M.R., (adhering to the practice of the Registrar General) and thus including its chance variation in about 14 out of 15 and 160 out of 161 cases respectively on the basis of an assumed normal distribution of the chance variations. According to the size of the random sample (more precisely, according to the number of deaths in the population group) these lines are at different distances from the mean line. Again adhering to the practice of the Registrar General, they represent the " probable " and " highly probable " border lines between chance variation and true or real differences in the S.M.R., according to sample size. They can also be regarded as a sort of mathematical gauge by which the

CONTROL CHART OF S.M.R., MALES, ALL CAUSES. PART I

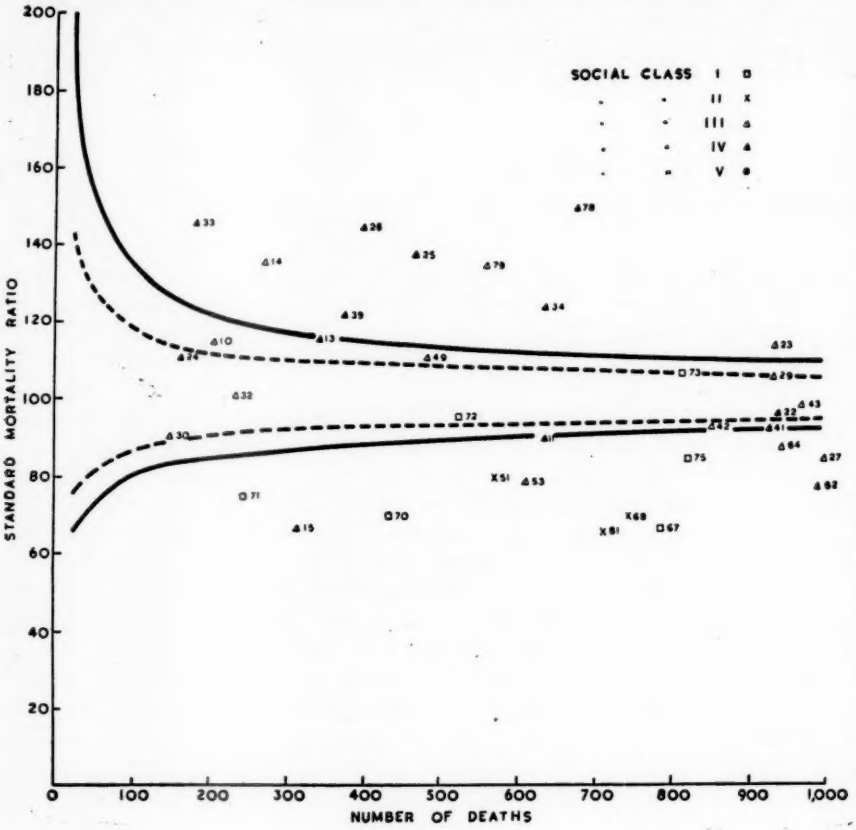

FIG. 1.

significance of a difference in S.M.R.s is quickly ascertained. All we have to do is to plot the S.M.R. for the occupation group in question as the vertical ordinate against the total number of deaths in that group during the time under consideration as the horizontal ordinate or abscissa for the cause or causes in question. If the point falls beyond the outer control line (2·5 times the standard error) we can be satisfied of a real difference and may use it as a reliable pointer to further investigation. If the point falls beyond the inner control limit (1·5 times the standard error) but not beyond the outer control limit, the difference is probably significant and well worth investigating further.

As an illustration of the method the following control charts have been prepared to show in males the S.M.R. for all causes (Figs. 1 and 2); influenza (Fig. 3); respiratory

TABLE I

KEY TO FIGS. 1 AND 2 GIVING LIST OF OCCUPATIONS FOR WHICH S.M.R. IS SIGNIFICANTLY HIGH AND LOW

High			Low		
Social Class	No.	Occupation	Social Class	No.	Occupation
I	—		I	67	Bank and insurance officials
				70	Clergymen (Anglican Church)
				71	Ministers of religion (excluding R.C.)
				75	Professional engineers
II	28	Employers and managers in Occupation Orders VIII-XXI, XXXI	II	1	Farmers and their relatives
				12	Employers and managers: gas, bricks, chemicals, etc.
	77	Inn-, hotel-keepers		51	Railway officials
				63	Commercial travellers
				64	Retail salesmen, grocery
				68	Civil Service and Local Authority: administrative, executive
				74	Teachers (not music)
				81	Draughtsmen, costing clerks, etc.
III	4	Coal hewers and getters	III	2	Gardeners, nurserymen, florists
	14	Potters, ware-makers, etc.		15	Workers in chemical processes
	16	Furnacemen, rollers and skilled assistants		27	Plumbers (not chemical plumbers)
	17	Metal moulders and die casters		35	Makers of textile goods
	23	Boiler-makers, platers and iron shipwrights		38	Bakers and pastrycooks
	46	Masons		40	Carpenters

tuberculosis (Fig. 4); cancer (Fig. 5).

For each chart the occupational groups have been listed for which the S.M.R. falls beyond the outer control limits, and thus signifies that the mortality, due to all causes or to the specified diseases, exceeds or is less than that of the total population by an amount which must be regarded as significant.

In this way we are not misled by the great number of S.M.R.s, differing from 100 by greater or smaller amounts, which very often mean nothing since they may be due to sampling fluctuations only. The comparison between

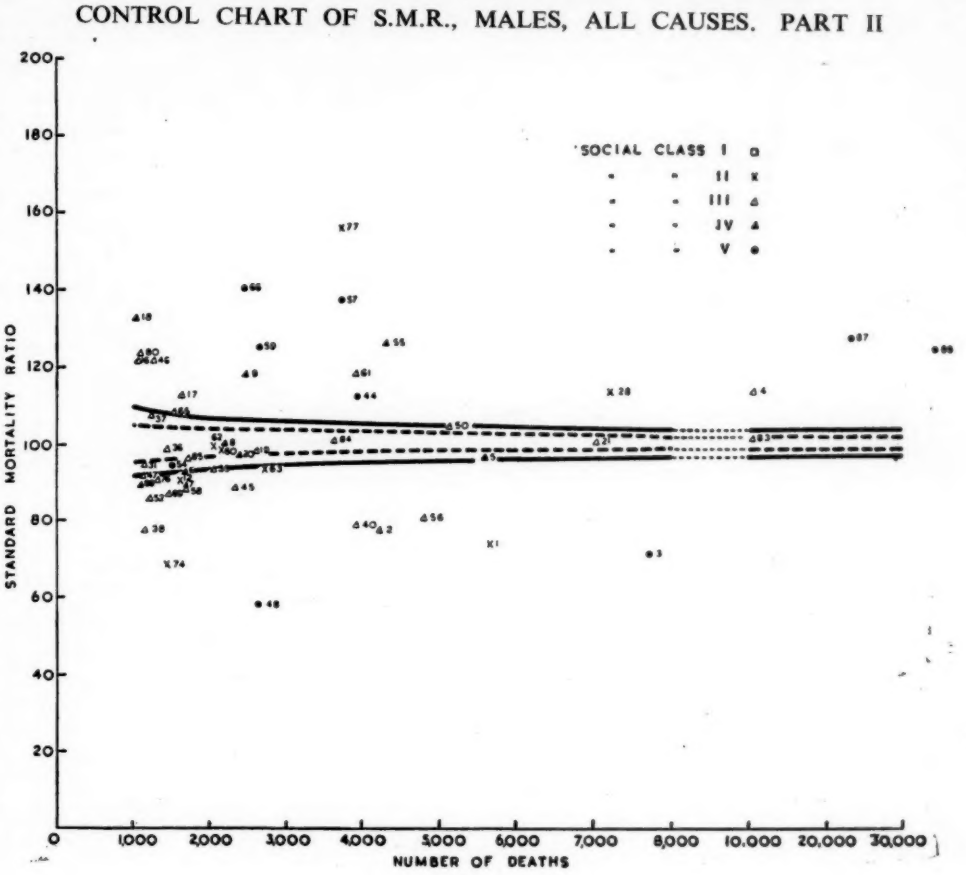

FIG. 2.

TABLE I—*Continued*

	61	Retail proprietors: dairy, meat, fish, greengrocery		45	Bricklayers
	65	Retail salesmen: dairy, meat, fish, greengrocery		47	Platelayers
	79	Waiters		52	Railways: engine drivers
	80	Hairdressers		53	Railways: signalmen
				56	Road transport: motor drivers
				58	Postmen and sorters
				69	Police
				76	Domestic servants (indoor)
				82	Typists and other clerks
IV	9	Coal mines: workers above ground	IV	6	Coal mines: conveying material to shaft
	18	Iron and steel foundry furnacemen		7	Coal mines: making and repairing roads
	25	Metal grinders		11	Stone miners, quarriers
	26	Metal glazers, polishers, etc.		86	Boiler firemen and stokers
	33	Textile strippers and grinders			
	34	Textile dyers			
	39	Makers of alcoholic drinks			
	55	Road transport: horse drivers			
	78	Barmen			
V	44	Builders, masons, and labourers	V	3	Agricultural and gardeners' labourers, etc.
	57	Water transport: dock labourers		48	Navvies in building trade, etc.
	59	Messengers and porters			
	66	Costermongers, newspaper sellers			
	87	General labourers			
	88	General labourers and other unskilled workers			

CONTROL CHART OF S.M.R., MALES, INFLUENZA

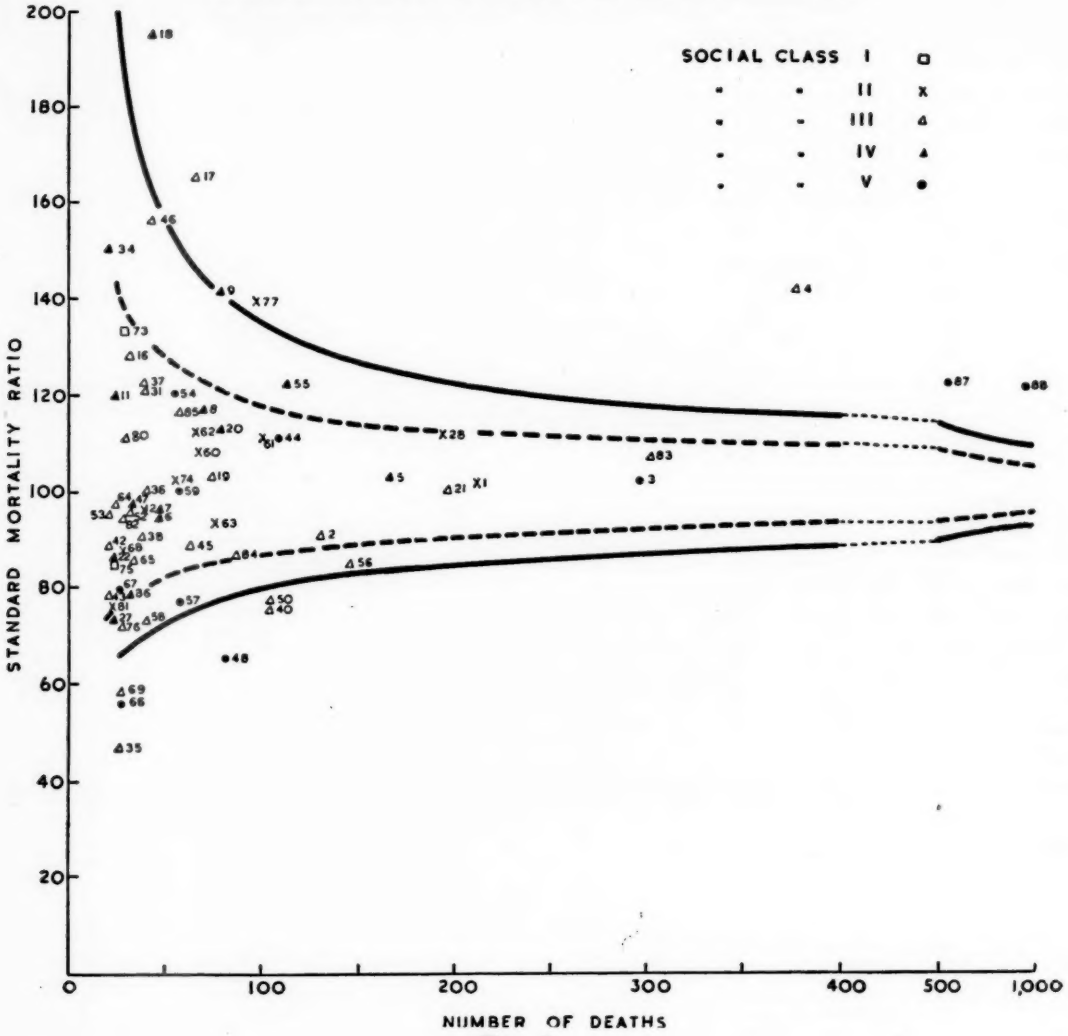

FIG. 3.

TABLE II

KEY TO FIG. 3 GIVING LIST OF OCCUPATIONS FOR WHICH S.M.R. IS SIGNIFICANTLY HIGH AND LOW

High			Low		
Social Class	No.	Occupation	Social Class	No.	Occupation
I	—		I	–	
II	77	Inn-, hotel-keepers	II	—	
III	4 17	Coal hewers and getters Metal moulders and die casters	III	35 40 50 69	Makers of textile goods Carpenters Paper hangers, painters, etc. Policemen
IV	18	Iron and steel foundry furnacemen	IV	—	
V	87 88	General labourers General labourers and other unskilled workers	V	48 66	Navvies in building trade, etc. Costermongers, etc., newspaper sellers

the graphs for influenza and respiratory tuberculosis is very instructive in this respect. Although even in the former the S.M.R.s often differ widely from one another and from 100, yet most of them are comprised between the outer control limits, thus affording no clue whether influenza is an occupational disease to a high degree.

Respiratory tuberculosis, on the other hand, shows a very great scatter of S.M.R.s, extending considerably beyond the outer control limits, and thus characterizing the disease as highly "occupational" in the wider sense of

CONTROL CHART OF S.M.R., MALES, RESPIRATORY TUBERCULOSIS

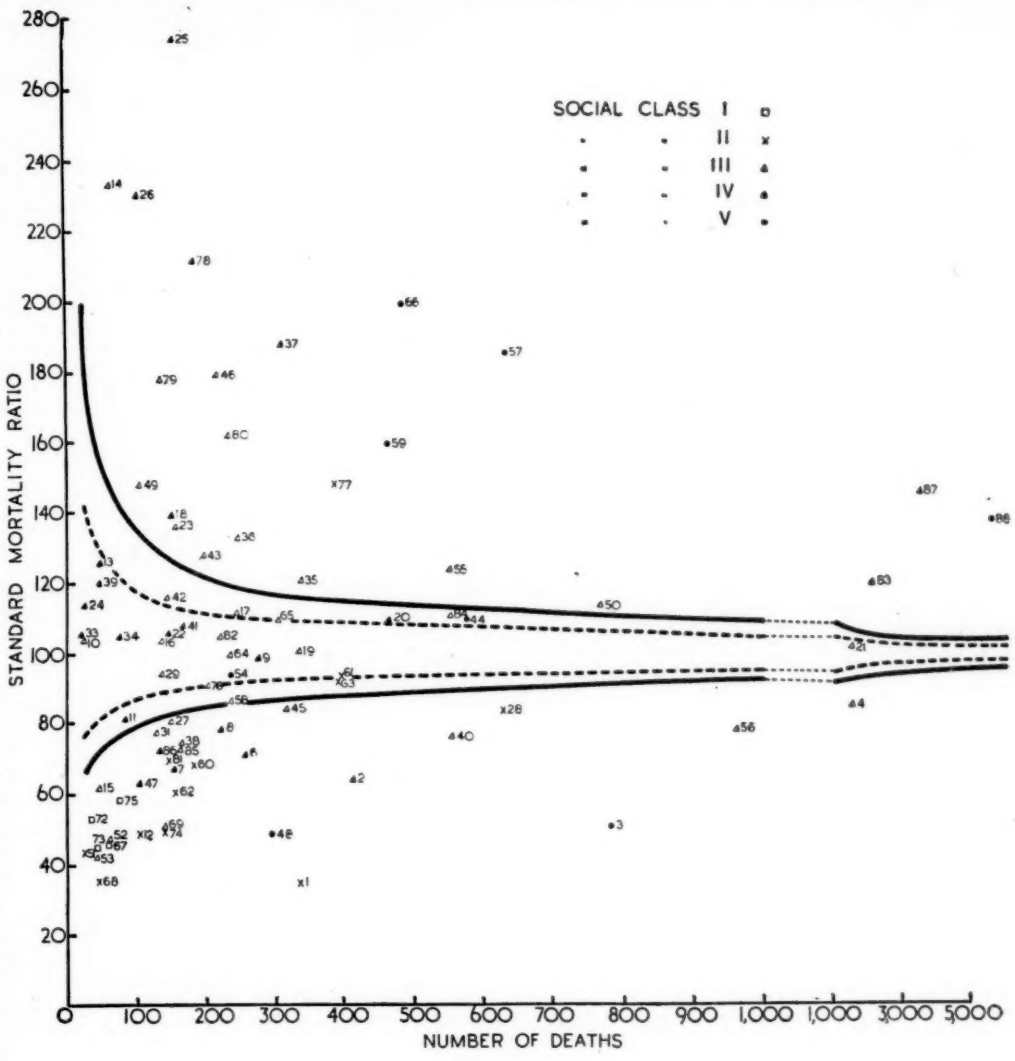

FIG. 4.

TABLE III

KEY TO FIG. 4 GIVING LIST OF OCCUPATIONS FOR WHICH S.M.R. IS SIGNIFICANTLY HIGH AND LOW

High			Low		
Social Class	No.	Occupation	Social Class	No.	Occupation
I	—		I	67	Bank and insurance officials
				72	Judges, barristers, solicitors
				73	Physicians, surgeons
				75	Professional engineers
II	77	Inn-, hotel-keepers	II	1	Farmers and their relatives
				12	Employers and managers: gas, bricks, chemicals, etc.
				28	Employers and managers in Occupation Orders VIII-XXI, XXXI
				51	Railway officials

TABLE III—*Continued*

				60	Retail proprietors, etc., e.g. grocery
				62	Wholesale proprietors, etc.
				68	Civil Service and Local Authority: administrative and executive
				74	Teachers (not music)
				81	Draughtsmen, costing clerks, etc.
III	14	Potters, ware-makers, etc.	III	2	Gardeners, nurserymen, florists
	35	Makers of textile goods		4	Coal hewers and getters
	36	Boot and shoe makers, repairers		15	Workers in chemical processes
	37	Boot and shoe workers and factory operatives		27	Plumbers (not chemical plumbers)
				31	Textile weavers (cotton)
	43	Printing machine minders, printers, etc.		38	Bakers and pastrycooks
	46	Masons		40	Carpenters
	49	French polishers		45	Bricklayers
	50	Paper hangers, painters, etc.		52	Railways: engine drivers
	55	Road transport: horse drivers		53	Railways: signalmen
	79	Waiters		56	Road transport: motor drivers
	80	Hairdressers		69	Police
	83	Typists and other clerks (other than Civil Service)		85	Stationary engine drivers not underground in mines
IV	18	Iron and steel foundry furnacemen	IV	6	Coal mines: conveying material to shaft
	25	Metal grinders		7	Coal mines: making and repairing roads
	26	Metal glazers, polishers, etc.		8	Coal mines: other workers below ground
	78	Barmen		47	Platelayers
	87	General labourers		86	Boiler firemen and stokers
V	23	Boiler-makers, platers and iron shipwrights	V	3	Agricultural and gardeners' labourers, etc.
	57	Water transport: dock labourers		48	Navvies in building trade, etc.
	59	Messengers and porters			
	66	Costermongers, newspaper sellers			
	88	General labourers and other unskilled workers			

TABLE IV

KEY TO FIG. 5 GIVING LIST OF OCCUPATIONS FOR WHICH S.M.R. IS SIGNIFICANTLY HIGH AND LOW

High			Low		
Social Class	No.	Occupation	Social Class	No.	Occupation
I	—		I	67	Bank and insurance officials
				70	Clergymen (Anglican Church)
				73	Physicians, surgeons, etc.
II	28	Employers and managers in Occupation Orders VIII-XXI, XXXI	II	1	Farmers and their relatives
				68	Civil Service and Local Authority: administrative, executive
	77	Inn-, hotel-keepers		74	Teachers (not music)
				81	Draughtsmen, costing clerks, etc.
III	16	Furnacemen, rollers and skilled assistants	III	2	Gardeners, nurserymen, florists
	17	Metal moulders and die casters		27	Plumbers (not chemical plumbers)
				40	Carpenters
				53	Railways: signalmen
				56	Road transport: motor drivers
IV	13	Skilled workers in gas works service	IV	5	Coal mines: workers below ground other than hewers
	18	Iron and steel foundry furnacemen			
	39	Makers of alcoholic drinks		6	Coal mines: conveying material to shaft
	55	Road transport: horse drivers		7	Coal mines: making and repairing roads
	78	Barmen		8	Coal mines: other workers below ground
V	44	Builders, masons, and labourers	V	3	Agricultural and gardeners' labourers, etc.
	57	Water transport: dock labourers		48	Navvies in building trade, etc.
	59	Messengers and porters			
	87	General labourers			
	88	General labourers and other unskilled workers			

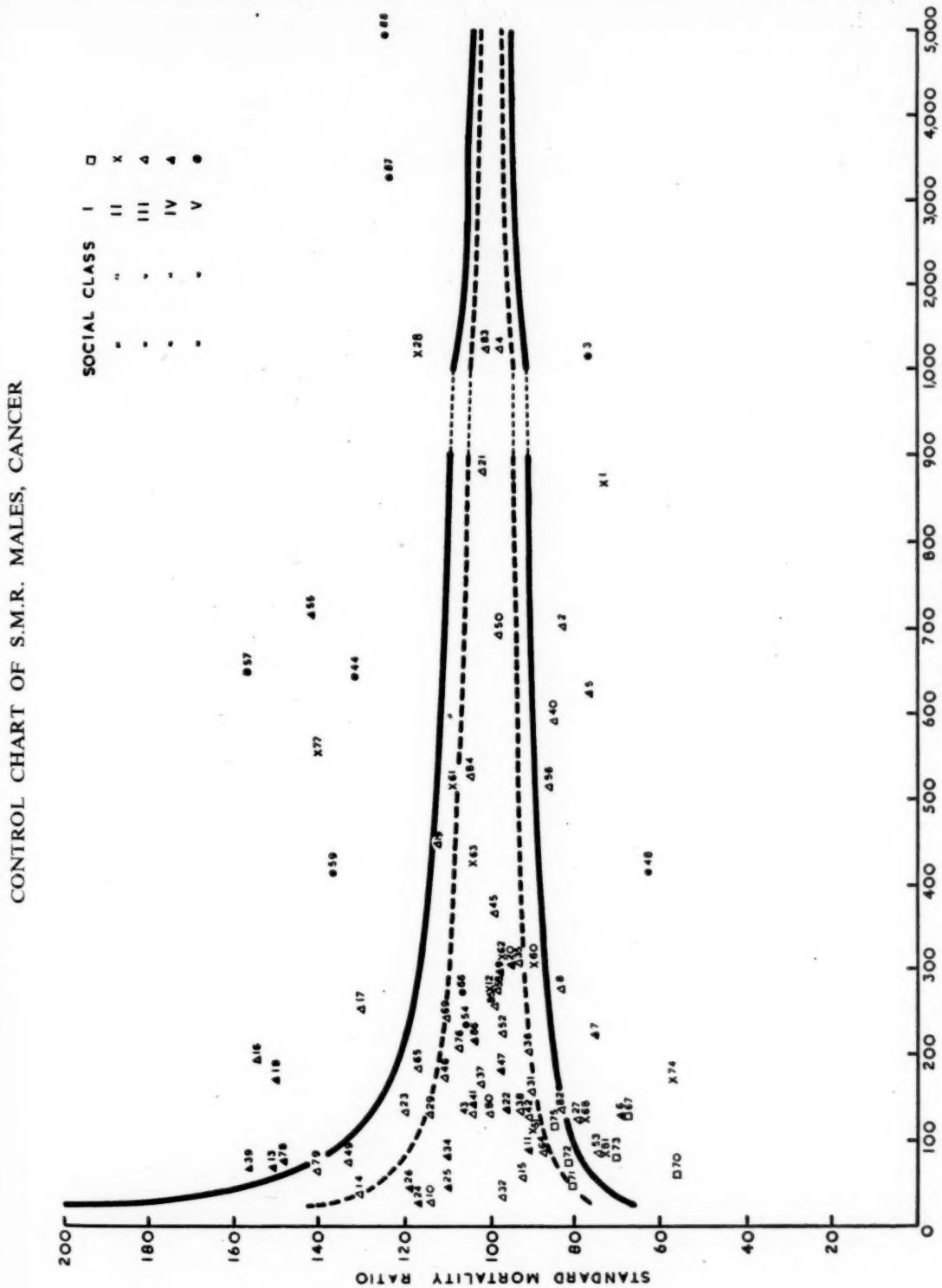
CONTROL CHART OF S.M.R. MALES, CANCER
SOCIAL CLASS I □
" II ×
" III △
" IV ▲
" V ●
STANDARD MORTALITY RATIO
NUMBER OF DEATHS
0
100
200
300
400
500
600
700
800
900
1,000
2,000
3,000
4,000
5,000
20
40
60
80
100
120
140
160
180
200

FIG. 5.

the term. That is, either the employees of certain occupational groups are at an increased risk of dying from respiratory tuberculosis, or a sort of selection takes place which makes persons afflicted by the disease gravitate to certain occupations more than to others.

On the basis of the control chart it is possible to express the scatter of the S.M.R. which is characteristic for a given disease in a single figure. One way of doing this is to count the points outside the outer control limits and express their number as a percentage of the total number of points plotted. We thus obtain for

Influenza	12/67 ..	18%
Cancer	32/86 ..	37·2%
Respiratory tuberculosis	57/84 ..	67·85%
All causes	60/88 ..	68·2%

For the purpose of comparison between different diseases, or between a specified disease and "all causes," index figures might easily be constructed. Taking for instance the percentage of points outside the control limits for "all causes" as our standard of comparison, i.e. as 100 per cent., the relative scatter for the three specified causes results as:—

Influenza, 26·5 or approximately one quarter of the scatter due to "all causes"; cancer, 54·7 or slightly more than one half of the scatter due to "all causes"; respiratory tuberculosis, 99·7 or approximately the same as the scatter for "all causes."

Although the Registrar General distinguishes by means of the S.M.R. between occupational groups with regard to a specific cause of death, it seems very desirable to have a method for distinguishing between different causes of death with regard to the extent to which they are selective in their action upon occupational groups. The integrating device which is needed for doing this is provided by the control chart, giving a complete picture and taking at the same time the standard error of the S.M.R. into account. It reveals the degree of significant heterogeneity in the action of a specified lethal agent upon the occupational groups. It also enables us to express that degree of heterogeneity, and the difference between different causes of death in that respect by a single index figure.

References

"The Registrar General's Decennial Supplement. England and Wales, 1931, Part IIA, Occupational Mortality." 1938. London.

Yule, G. N. (1934). *J.R.S.S.*, XCVII., I., 29.

Herdan, G. (1948). "Quality Control by Statistical Methods." Edinburgh.

Brit. J. soc. Med. (1949), **3**, 77-84.

SOCIAL MEDICINE

A STUDY OF THE FIRST HUNDRED PATIENTS OF A UNIVERSITY DEPARTMENT OF INDUSTRIAL HEALTH

BY

R. C. BROWNE, I. F. BECK, and R. I. McCALLUM

(*From the Nuffield Department of Industrial Health, University of Durham, King's College, Newcastle-upon-Tyne*)

The first report of the Nuffield Foundation (1946), referring to the subject of industrial health, states that:

> Industrial hazards and diseases, which hitherto have claimed most attention, do not constitute the whole of the subject. . . . departments of industrial health should be closely allied with whatever organization the universities should establish for the study and teaching of social medicine. Industrial health is one of the most potent, if still mainly potential, executive branches of social medicine.

It is timely now to ask what kind of problems are encountered in the routine practice of a department of industrial health in an area of heavy industry; how does it deal with them, and how does the practice compare with the precepts already laid down (Browne, 1947).

METHODS

When clinical work was started a circular was sent out under the joint auspices of the university and its teaching hospital to all general practitioners in the North-East Hospital Region. This drew their attention to the formation of the department, and pointed out that it would receive cases of medical industrial disease (with the exception of skin conditions) scheduled under the Workmen's Compensation Acts, together with any other medical conditions in the causation of which the nature of the patient's work was thought to have played a part. It was difficult to forecast the response to this circular, but it was thought wiser to aim at one which would be too small rather than too big, hence the rather narrow drafting of the notice.

An out-patient clinic was instituted in a suite of three communicating rooms, two for the clinicians who see patients simultaneously, and the third for the social assistant. The patients see the doctor first and then the social worker, because they expect to start by telling a doctor about their symptoms, and until they have done this they are not ready to discuss their social difficulties. Also, the social worker would have to take her social history blindfold if she had no indication of the clinical and industrial histories and their possible implications. The clinical and industrial notes and the social summary were designed, and are kept, as a whole inside one folder. Letters are written to the patients' doctors by the clinician and have references to these three divisions of the whole problem. Management is decided jointly between clinician and social worker, and since both are members of the same department, formal case conferences are unnecessary.

The same principles guide the in-patient work, which is done in the Department's own beds within the main division of medicine, with which there is the closest connexion.

Full details of a single case history will serve to illustrate the scope of the information recorded.

> Mr. A.B., aged 34 years, had noticed for the last three weeks increasing weakness of the right hand, which had become stiff, clumsy, and useless. He had had " stomach cramps " and constipation for one week.
>
> For the last 20 months he had worked as a plate paster in a lead battery works. He had also been weighing dry red lead powder and then mixing it with distilled water and sulphuric acid to form a paste. His previous occupations were: age 14-28, plasterer's labourer; age 28-32, in Army (Royal Engineers).
>
> Physical examination showed him to be a pale, thin man, with a bilateral congenital ptosis more marked on the right. Wasting and weakness of the muscles of the right hand, forearm, arm, and shoulder were observed, and right wrist drop. Sensation was absent over the right hand and forearm, with " evening glove " distribution present over the arm. Examination disclosed no other abnormality. Blood examination gave a red cell count of 3·95 mill. per cu.mm.; Hb. 71 per cent.; colour index: 0·9.
>
> His social history was that he was born in Newcastle-upon-Tyne, son of a regimental sergeant major who

became under-manager of a lead works. After marrying at 24, he shared a house with his cousins and later with his in-laws, with whom he is still living together with his wife and three children. His employer's comment was that he was one of his best workmen.

He had been earning up to £7 10s. per week until he became ill, but as soon as he stopped work his wage ceased. Sickness benefit was not paid, as there was a question of workmen's compensation. His wife had to apply for Public Assistance until the compensation claim was settled. He was a nervous man, unsure of himself and easily upset by any suggestion that he might be malingering. Controversy over compensation and repeated examinations by different doctors in this connexion made him increasingly introspective and preoccupied with his illness.

The patient's factory was visited; it employed only six men, and was established during the 1939-45 war in old accommodation. His job was to spread a red lead and sulphuric acid paste on to a solid lead grid about 9 in. square. Rows of pasted grids were wrapped in paper and taken into the "drying room," which was merely an area of the main workshop in which there was a coke stove. When dry, each plate was broken in half to form two small ones. The plates were then packed in boxes. In a corner was a small milling machine for trimming the plate lugs, and although this was under an exhaust ventilation hood, a quantity of the metallic lead dust came out into the main workshop. The workshop was dry-swept, and the red dust of lead oxide could be seen lying on the lampshades and ventilators. On coming to work in the morning, a man took his dusty overalls out of a locker and put in his coat, thereby transferring lead oxide dust from his work to his home clothes.

The clinical diagnosis was, therefore, lead poisoning with hysterical anaesthesia of the right arm. The social diagnosis was loss of earnings, change of employment, and compensation difficulties, and the industrial diagnosis a slum factory with inadequate management and hygiene.

The patient was managed by treatment with bed rest for two weeks, followed by one week's retraining. A high calcium diet with vitamin D and ferrous sulphate was given. The right wrist was splinted in the "cock-up" position, and then increasingly active exercises were given to the affected hand and arm.

While he was in hospital, the Disablement Rehabilitation Officer from the local employment exchange was asked to see him, as the risk of further contact with lead made it unwise for him to return to any job in the firm where he had previously worked. Before he started work legal advice about his compensation was obtained. He was then found work by the Ministry of Labour in a clothing factory. Here he took messages, swept the floor, and tied up parcels, and with wages and partial compensation his income was £5 10s. per week.

He was receiving compensation under the old Workmen's Compensation Act, and after six months the insurance company raised the question of a lump sum settlement. The complications which followed were most perplexing to the patient, and no final settlement was made. Weekly payments were continued for 11 months, and then discontinued on the grounds that the disability was no longer the result of lead poisoning. The patient's income was thus reduced to £4 per week. He still maintained that he could not use his hand for heavy manual work, and he had been obliged to refuse an attractive and better paid job for this reason. He felt that he had been unjustly treated, and decided to take the case to court. The final results of this unfortunate medico-legal controversy remain to be seen. Meanwhile, the patient is being encouraged to take a practical and unemotional view of his difficulties.

During the earlier stages of his illness he needed a good deal of help from the social worker, but the support which was given to him then was gradually withdrawn. As his self-confidence increased, he became able to take the initiative in dealing with problems for himself, and his morale has improved during the last few months.

Two unofficial visits to the patient's former factory by the departmental staff and an official visit from the factory inspector, led to improvements in the level of hygiene. These were first shown by improvements in the clothes locker system, together with greater attention to floor damping and cleanliness.

Occupational Groups

Sixty-nine per cent. of the patients were coal-miners, usually coal face workers or men who had been face workers, but for various reasons, often dyspnoea, had changed to less strenuous odd jobbing work paid at a daily rate. One patient had mined lead, and another ironstone. Four men worked in foundries, and there were two arc welders and three lead workers (one pasting batteries, and two smelters). There were also four tar workers. The remaining 16 men were engaged in various occupations which ranged from baker to bus conductor, and included a trinitrotoluene worker, a plumber's mate, a steel erector, a coke worker, and a phenol worker (Table I).

TABLE I

Analysis of Occupations

Occupation		No. of Cases
Mining		
Coal	69	
Lead	1	
Ironstone	1	
	—	71
Lead workers		3
Tar workers		4
Foundry men		4
Arc welders		2
Various trades		16
		100

TABLE II

ANALYSIS OF TYPES OF ILLNESS IN THE SERIES

	Type of Illness		No. of Cases	Totals
Chest	Pneumoconiosis of coal miners (1 with pulmonary tuberculosis and 1 with miners' nystagmus)		21	
	Siderosis		2	
	Silicosis (1 with pulmonary tuberculosis)		2	
	Pulmonary tuberculosis		5	
	Chronic bronchitis and emphysema		23	
	Other chest conditions		4	57
	Pulmonary oedema, following shot firing	1		
	Carcinoma of bronchus	1		
	Spasmodic asthma	1		
	Acute bronchiolitis, following exposure to metal fume	1		
Intoxications	Trinitrotoluene		1	
	Lead		3	
	Carbon monoxide (9 industrial, 2 other)		11	15
Dermatological	Tar (2 dermatitis, 2 epitheliomas)		4	
	Various		2	6
Miscellaneous	Psychiatric		7	
	Cardiovascular		6	
	Miners' nystagmus		2	
	Various conditions		7	22
				100

MEDICAL PATHOLOGY

Diseases of the chest were found in 57 per cent. of the men (Table II). Of these, pneumoconiosis of coal miners (21 per cent.) and chronic bronchitis (23 per cent.) were by far the commonest conditions diagnosed. All the cases of coal miners' pneumoconiosis were in coal face workers, and the radiological changes which they presented are similar to those described by Hart and Aslett (1942) and more recently by Davies and Mann (1948), whose method of classification has been used. Sixteen men had simple and five complicated pneumoconiosis (Table III). Two cases of silicosis were seen, one of the nodular type in a steel dresser, and the other in a lead miner with complicating open tuberculosis. In both these cases the diagnosis was confirmed at autopsy. There were also two arc welders in whom there were radiological changes suggesting siderosis. In 23 men there was a diagnosis of chronic bronchitis and emphysema, with varying degrees of disability. These were also mostly face workers in coal mines who had been referred to us primarily to exclude pneumoconiosis. Their average age was 54 years, compared with an average age of 51 years in those with pneumoconiosis. Of other chest conditions seen, there were five cases of pulmonary tuberculosis without any occupational hazard complicating it, and one case each of bronchial carcinoma, spasmodic asthma, acute bronchiolitis following exposure to metal fume, and pulmonary oedema following shot firing.

TABLE III

PNEUMOCONIOSIS OF COAL MINERS (CLASSIFIED ACCORDING TO DAVIES AND MANN, 1948)

Type	Category	No. of Cases
Simple	1	6
	2	3
	3	7
	4	0
Complicated	A	0
	B	3
	C	1
	D	1
		21

Industrial poisoning, mainly by carbon monoxide, occurred in 13 patients, eight of whom were miners admitted following underground explosions, and one a man who had been working on a gas main. Lead intoxication was found in three men, two of

whom were engaged in smelting, and the third in battery pasting. One man, a T.N.T. worker, was thought to have a toxic gastritis. There were no fatal results in all these men.

We have not set out to treat skin conditions, but a few (six cases) have been seen, including two of epitheliomata in tar workers. A group of seven men in whom the main complaint appeared to be a psychological disorder, were included in the first 100 patients seen, and were reassured or referred elsewhere for specialist treatment.

Six patients were found to have disorders of the cardiovascular system, all coal miners except one, and all elderly apart from a miner of 35 years with mitral stenosis. Miners' nystagmus was seen in three men, one of whom had a pneumoconiosis for which he had been fully compensated, and whose capacity for work was greatly limited by that alone.

Finally, there were seven men in whom various conditions were found: chrome ulceration of the nasal septum, chronic nephritis, blepharitis, chronic naso-pharyngitis, laryngeal fibroma (in each case unrelated to occupation), a resettlement problem, and one fit man who had been suspected of having pneumoconiosis.

Social Pathology

The most frequent clinical conditions seen were chronic bronchitis, coal miners' pneumoconiosis, and cases of gassing, and it is of value, therefore, to trace the relation of social to medical pathology in those conditions.

The commonest and most important social problems found among adult patients are material difficulties connected with employment, income, housing, and domestic care, and the emotional difficulties which may complicate them (Beck, 1947). The number and severity of these difficulties among any group of patients depends on the length of the illness and the amount of disability which it causes. A clear-cut illustration of this may be seen in Table IV, where the patients with diseases of the chest and those with carbon monoxide or other gas poisoning have been separated from the rest of the series. The patients with chest diseases were not acutely ill, few of them could benefit from medical treatment, and many of them seem from a narrow clinical viewpoint to be rather uninteresting. But from a social point of view their pathology was sometimes acute and often highly complex, and they formed an unusually interesting group. The men who had been overcome by gas poisoning were very ill and were in urgent need of skilled medical treatment, but they made rapid and complete recoveries. Their social difficulties were minimal, and no social treatment was needed. The only exception to this was an attempted suicide whose social problems had been the cause, and not the result, of his carbon monoxide poisoning.

TABLE IV*

Effects of Illness on Social Background

Disease	Degree of Social Difficulty				
	None	Minor	Serious	Unknown	Total
Pulmonary ..	3	10	39	3	55
Gas poisoning ..	10	2	1	0	13
Other	8	8	13	3	32
Totals	21	20	53	6	100

* In this and later tables one patient with bronchiolitis and one with pulmonary oedema, both of whom were overcome by fumes while working, have been included in the gas poisoning group, and not among diseases of the chest.

Problems have been classed as serious when some drastic alteration of the social background was caused by the illness or became necessary as a result of it, or when help was needed to avert hardship and anxiety. When the social impact of illness can be met without assistance and does not involve difficult readjustments for the patient or his family, any problems which are present can be classed as minor ones. Thus, changing his job is a serious matter to a patient if he cannot find suitable work without help, or if the change means a big drop in income. If he is able to find a new job himself at a rate which compares reasonably well with what he got before, this is regarded as a minor problem. In the present series of men, most of whom were earning high wages, the economic effects of illness were not rated as serious unless income was reduced by more than 25 per cent. Pre-existing social problems which might have precipitated illness or complicated its results were so uncommon that they have not been listed separately.

Most of the patients were wage earners with family responsibilities, and the need to change or to cease work often caused a complete dislocation of their own and their families' lives. Table V, which shows what happened to the men as workers, gives little more than a hint of what it meant to them as complete human beings. Less than 40 per cent. of the whole group were able to return to their original jobs, and scarcely more than 20 per cent. of those with diseases of the chest were able to do so. Those who changed their jobs were better off than those who remained idle, but most of them had their income permanently reduced. For the men

with chest diseases, most of whom were coal face workers, the change usually involved a weekly drop in income from £8 or more to £5 or less. To men in their forties and fifties, who had worked hard for a mere pittance during the lean years, it came as a particularly hard blow to have to give up their work now when it was well paid. Nor was the economic hardship the only one, for the change often resulted in a serious waste of human material. One man of unusual intelligence and ability, who had been hoping to send one of his children to a university, had worked underground as a deputy, a highly responsible post. He could get no work at all through the Ministry of Labour, and ended up by making clay plugs for shot firing, a type of work suited to the lowest grade of manual worker.

TABLE V

EFFECT OF ILLNESS ON WORK: TOTAL SERIES

Disease	Work			Retired	Died	Unknown	Total
	Same	Changed	Idle				
Chest	12	15	19	3	3	3	55
Gas poisoning ..	12	1	0	0	0	0	13
Others	11	11	4	3	0	3	32
Totals	35	27	23	6	3	6	100

The more detailed analysis in Table VI suggests that men with chronic bronchitis are better off than those with pneumoconiosis as regards getting back to work. Although the figures are small, they illustrate certain differences. Men who had been certified as disabled by pneumoconiosis were legally debarred from returning to underground work, or indeed to any work around the mine apart from a few non-dusty surface jobs, and these often required more physical effort than some of the jobs away from the coal face underground. The only men with pneumoconiosis in this series who remained in their old jobs were two who preferred not to take the economic risk involved in making a claim for compensation, and one who was certified as having the disease, but not disabled by it. In such cases, a man was allowed to choose whether or not he would accept the verdict. If he accepted, he would get half wages for a limited time, but would be debarred from going back to mining, although not entitled to any further compensation. This man's decision to continue at work underground seemed sensible enough in the circumstances, but it is worth noting that his union advised him most strongly against it, and only a determined and level-headed man would have persisted in such a course. The question of changing their work did not arise for men with chronic bronchitis with similar symptoms, and those who were partly disabled could often get less strenuous work below ground. This is reflected in the larger number of men with chronic bronchitis (Table VI) who were able to get different work without changing their employer. Under the new regulations for pneumoconiosis compensation, a different policy about suspension from mining may level out some of these differences in the future.

TABLE VI

EFFECT OF CHEST DISEASE ON EMPLOYMENT

Disease	Employment				Retired	Died	Unknown	Total
	Same	Changed		Idle				
		Same Employer	Different Employer					
Pneumoconiosis	3	2	4	10	1	0	1	21
Chronic bronchitis ..	6	9	0	5	2	1	0	23
Other chest conditions ..	3	0	0	4	0	2	2	11
Totals ..	12	11	4	19	3	3	3	55

For the miners who could not continue in underground work, the situation was similar whether they had pneumoconiosis or chronic bronchitis. If anything, the bronchitics might be worse off, since some priority was given to " compensation cases " in allocating the few available surface jobs. When the colliery had nothing to offer, the prospects of getting back to work were poor. These men were considered too old to be accepted for training by the Ministry of Labour, even if they could have been placed in their own districts when trained. Besides, mining was a way of life to most of them, so that the thought of leaving it struck at the very roots of their communal existence. In any case, the plain fact was that alternative work could seldom be found in the districts from which they came. One chronic bronchitic had already been on the books of the Ministry of Labour for three years, and he was an intelligent and stable man who was fit for any form of reasonably light work.

One of the four men with pneumoconiosis who got work with other employers was the deputy mentioned above who was employed in making clay plugs, and one man was able to get work as a night watchman. The two others were younger men of unusual initiative, one of whom lived within travelling distance of a trading estate and found work there, and the other was taken on by his local council as a park and beach attendant. The last

appointment may not have been altogether wise, as the duties included life-saving on a part of the coast where bathers often get into difficulties, but the patient managed his first summer season satisfactorily and hopes later to be relieved of this part of his responsibilities.

None of the patients with gas poisoning needed to change his job, except for the attempted suicide. Among those with other diseases who needed to change was a group of three men with lead poisoning and one with T.N.T. poisoning. Only one of these, a leadworker with severe wrist drop, had much difficulty in finding suitable work, and the final effect on income of changing their jobs was not serious for any of them. In the whole series, no patient who was physically and mentally fit for work failed to get a job, except in the groups with pneumoconiosis or chronic bronchitis.

Seventeen out of the 27 men who changed their jobs did so successfully by their own efforts. Seven more were able to get suitable work after we had approached their employers, and three had jobs found for them by the Ministry of Labour. Nine other men had been referred to the Ministry of Labour for resettlement, but five of these are still idle, and the other four eventually found jobs for themselves. Ten patients with pneumoconiosis and five with chronic bronchitis are still not working at a period from nine months to two years after they were first seen. Special attempts were made to find work for three of the men with pneumoconiosis, and three others were well enough to be signed on at the employment exchange, although they had no hope of getting work. Unsuccessful attempts were also made to find work for four of the bronchitics. Two men with pneumoconiosis and one with chronic bronchitis were probably unfit for anything except some form of home occupation. Two other men with pneumoconiosis were primarily disabled by other conditions, one by miner's nystagmus and the other by long-standing peptic ulceration.

There was not much to choose between the unemployed men with pneumoconiosis and those with chronic bronchitis as regards fitness for work, but the tendency was for the bronchitics to be rather worse off in this respect, and the social results were often more serious.

The average weekly income of the men who were unable to return to work was about £2. Individual amounts might be as little as 10s. 6d. a week for a man whose national health insurance payments had been reduced. They might be as much as £3 or over for men who were drawing unemployment benefit and partial compensation, or whose sick benefit was being supplemented by public assistance. As even full compensation at that time was little over £2 per week, the advantage of the men who could be compensated over those who could not was small. Under the new rates of insurance and compensation, both classes of men will benefit, but the economic difference between being disabled by pneumoconiosis or by chronic bronchitis will become more marked. In certain circumstances a man compensated for total disablement by pneumoconiosis under the new Act might get as much as he would in a light job, but the man with chronic bronchitis will only get half this amount.

Throughout the present series, men avoided application for public assistance whenever this was humanly possible, and when savings were exhausted after long absence from work, contributions from children, the earnings of wives, or help from relatives often tided the family over. A drastically lowered standard of living and the sacrifices of his family often added to the patient's depression and feeling of uselessness. It was this sense of being " on the scrap heap " and of no further use either to their families or to the community, which broke the men's spirits and caused the greatest suffering to all concerned. The men with bronchitis in the present series were affected in this way more seriously than those with pneumoconiosis.

Contrary to what might have been expected, not one of the patients with pneumoconiosis became preoccupied with compensation. The legal aspects of their illness caused some disturbance in two of the men with lead poisoning, but this did not seriously hinder their return to work. More serious compensation disturbances were found among unstable patients who had not got industrial diseases carrying compensation, but who persisted in blaming the work for their illness. Two men in particular illustrated the unfortunate results which well-meaning people, doctors as well as laymen, may produce by encouraging such an attitude. One of these men was an arc welder with siderosis, and the other operated a grinder in a machine shop and now has pulmonary tuberculosis. Neither had valid ground for claiming compensation, but both had been encouraged to embark on litigation and to fight losing battles until they had become so obsessed with their grievances that they were permanently unemployable.

In the present series, which contained a high proportion of intelligent and stable men, compensation claims had no adverse emotional effects, provided that they could be settled quickly and definitely. It was noticed that a successful claim often improved morale without removing the incentive to get back to work. Men seemed to feel

that any implication of personal failure in becoming ill was removed if responsibility for their breakdown was admitted by the industry, and they also felt that to some extent at least justice had been done. The men with chronic bronchitis often believed that their trouble must really be caused by the dust, and they felt that they were unjustly treated because they could not claim compensation, or continue to receive free coal, or remain indefinitely in their tied colliery houses, as the men with pneumoconiosis normally could. Emotional disturbances which resulted from physical incapacity and were severe enough to affect the men's behaviour and to throw added strain on their wives and families were seen in five men with chronic bronchitis and one with pneumoconiosis. It is a tribute to the tough moral fibre of the group as a whole that these were the only men affected to such a marked degree. The most impressive finding was the resilience with which so many men disabled by one or other of these diseases made the best of extremely unpleasant situations. Whenever it was possible to help them we felt that they were particularly well worth helping because they did so much to help themselves.

Twenty per cent. of the patients needed casework, and this usually involved one or more visits to their homes, and sometimes visits to employers as well. Fifteen per cent. needed simpler forms of help, but the rest could manage their own social and industrial affairs, and only needed advice and information.

Discussion

The main medical and social problem in this hundred patients is the emphysematous and dyspnoeic coal miner, with pneumoconiosis and chronic bronchitis as the chief associates of the emphysema. Acute carbon monoxide poisoning and psychological disease form two equally common groups. The three cases of lead poisoning and the single case of chrome ulceration of the nasal septum related in each case to small factories (which were visited) with low standards of environmental hygiene. A diagnosis of pneumoconiosis in a man who was not severely disabled was more serious socially than one of chronic bronchitis, because such men were legally prevented from working underground or in any work in coal mining, apart from a few non-dusty, surface jobs. This often meant forced unemployment because in a pit village mining is the only available work. More elastic legislation combined with the establishment and dispersal of light employment (other than coal mining) in the villages, as well as in any new towns which may be built, will considerably lessen the social effects of chronic chest disease in coal miners in this area. Both the medical and social results of industrial illness differ only in points of detail from other medical diseases, and successful management, as opposed to the narrower conception of "treatment," stands or falls with the unity of the medical and social team, and with the success of this team in securing the co-operation of the patient and of his home environment.

The amount of work involved in social management depends not so much on the severity of the problems as on their complexity. A single difficulty can often be dealt with by simple means. Reference to public assistance (or the Assistance Board as it would be now), a letter to an employer, even a full discussion with the patient and some advice or information, may be all that is needed to prevent serious trouble. In a complex problem a number of separate difficulties react on one another in such a way that the treatment of any one of them must take into account its effect on all the others. It is not sufficient here to refer the patient to one or more of the official bodies concerned with particular forms of help, such as financial assistance or rehabilitation. If there is a chance of improving the situation, it can only be done by the individual approach and intensive methods of social casework.

Close integration of clinical and social work made it possible for the patient's management always to be considered as a whole, and there was no risk that the patient would be given conflicting advice by clinician and social worker. It was much more difficult to continue this integration outside the hospital walls. However suitable the plan of action which clinician and social worker helped the patient to evolve, it might miscarry from the start unless it also had the approval of the patient's family, his employer, his trade union officials, and his private doctor. To secure this was a task which sometimes required much time and patience.

To compare the clinical practice with the precept (Browne, 1947), less home visiting has been necessary than was originally visualized, and when assessing physical hazards to health it has been found better that the clinician should do any industrial field visits either himself, or with the social worker, rather than that the latter, with her essentially non-technical background, should go alone. She does, however, visit employers independently to discuss with them the resettlement of patients whose working conditions are already known. No difficulties have been encountered in visiting either homes or works, and this may be ascribed to the fact that the visitors are representatives of an independent university department and have a therapeutic relationship with the patient.

SUMMARY

The medical, social, and industrial problems of the first hundred patients of a university department of industrial health are described.

Sixty-nine per cent. of the patients were coal miners, and the remaining 31 per cent. covered a wide range of occupations.

One-third of the coal miners seen had chronic bronchitis and emphysema, and about another third had pneumoconiosis. Carbon monoxide poisoning and psychological abnormality were the next commonest diagnoses.

The social pathology varied directly with the length of the illness and with the disability caused. Loss of earnings, together with the difficulty of obtaining lighter work, were the two most important social findings.

REFERENCES

The Nuffield Foundation: First Report (1946), p. 16 Oxford

Browne, R. C. (1947) *Brit. med. J.*, **1**, 838.

Davies, I., and Mann, K. J. (1948). *Proc. Ninth International Congress on Industrial Medicine* (to be published).

Beck, I. F., Gardner, F. V., and Witts, L. J. (1947). *Brit. J. soc. Med.*, **1**, 197.

Brit. J. soc. Med. (1949), **3**, 85-94.

MORTALITY AMONGST BABIES FROM INJURY AT BIRTH

BY

W. T. RUSSELL and IAN SUTHERLAND

From the Institute of Social Medicine, Oxford

Much has been said and written about infant mortality and particularly about its decline. Even statesmen have cited its decrease as suggestive evidence of the benefits which arise from their stewardship. They are, it would seem, oblivious of the fact that the decline is not of recent occurrence but one which has been operating over a period of many years. The facts are easy to appreciate. Fifty years ago the mortality in infancy in England and Wales exceeded 150 per 1,000 births; in 1948 it was 34, an impressive reduction which was particularly observable in the age group 3-12 months. The reduction in the first four weeks of life has been less satisfactory.

It may well be asked whether each contributory cause of death in early infancy has declined equally or whether some have declined more than others. If the average annual change in the mortality "under 4 weeks" during the period 1931-38 is expressed as a percentage of the mean death rate in that period in England and Wales, the statistical picture for certain broad categories is as in Table I.

It will be noted that in four instances the mortality has declined, the annual decrement being largest (9·1 per cent.) for convulsions. The death rate in the two remaining groups, congenital malformations and injury at birth, increased, and the increment for the latter was the greater as the values were 1·1 per cent. and 2·6 per cent. respectively. It should be stated, however, that there is a school of expert medical opinion which regards such observed increments as fictitious. Although it recognizes the total neonatal mortality as a reliable figure, it suggests that the constituent causes defy statistical analysis owing to the inability of doctors in general to state, with any degree of precision, the exact cause of death at this period of life. The difficulties of accurate certification of causes of death in the first four weeks of life are well recognized, but it would seem unlikely that medical men are in the habit of exaggerating birth injury as a cause. Indeed, the joint report of the Royal College of Obstetricians and Gynaecologists and the British Paediatric Association (1949) has suggested a much higher rate of mortality from birth injury than is revealed by the Registrar General's tabulations.

It is not the province of a statistician to question the validity of medical diagnosis. In the absence of a sudden or startling departure from the normal mortality (apart from epidemic experience), he accepts the declared findings as facts although he is, at the same time, fully conscious of the vagaries of medical opinion and the influence of new precisions on diagnosis. We have been told, however, that the increments which have been demonstrated in the mortality from injury at birth may possibly be due to a transfer of deaths from asphyxia, atelectasis, or convulsions. The death rate from convulsions has declined, and convulsions in children under four weeks are believed, in many instances, to be the aftermath of an injury during delivery. As a test we calculated the mortality of a combined group of causes of death, namely convulsions, injury at birth, asphyxia, and atelectasis, over a

TABLE I

AVERAGE ANNUAL CHANGE IN MORTALITY "UNDER FOUR WEEKS" EXPRESSED AS PERCENTAGE OF MEAN DEATH RATE

	Convulsions	Congenital malformation	Congenital debility	Premature birth	Injury at birth	Other causes
Mean death rate under 4 weeks per 1,000 live births	0·89	3·65	1·52	16·02	2·30	6·28
Percentage annual increase or decrease	−9·1	+1·1	−6·7	−1·9	+2·6	−1·3

period of years. In 1945 approximately 4,000 deaths of babies under one month were ascribed to this causal group. If it can be shown that the mortality from this combination has increased, then there is little basis for suggesting that a transference of deaths affords an adequate explanation. The death rates at ages under four weeks per 1,000 births were as in Table II for (A) the combined group, and (B) the residual neo-natal mortality.

TABLE II

DEATH RATES UNDER FOUR WEEKS PER 1,000 BIRTHS
A. COMBINED B. RESIDUAL

Period	A	B
1921-30	5·03	27·53
1931-39	5·20	25·48
1945	5·55	19·21

It will be seen that the death rates in (A) differ from those in (B) in their movement; if anything, they indicate a slight increase in their secular trend. In this connexion a comment by Martin is relevant:

A matter for concern is the steady rise in deaths due to injury at birth, from 1·22 in 1920 to 2·59 in 1938. A similar trend has been shown by the United States where the mortality from injury at birth rose from 3·9 in 1920 to a peak of 5·3 in 1930 and then declined slightly to 4·8 in 1939.

It is not possible to obtain an inclusive rate for the three causes of death in the main geographical regions of England and Wales over a long series of years, as deaths from asphyxia and atelectasis were not published separately for each area until 1940. Accordingly the combined rate for convulsions and injury at birth under four weeks was calculated and is set out in Table III.

TABLE III

COMBINED DEATH RATE FOR CONVULSIONS AND INJURY AT BIRTH

Area	Average death rate in period 1931-45
Greater London	2·48
South-Eastern Counties (outside Greater London)	2·52
North I	3·97
North II	3·41
North III	3·35
North IV	3·46
Midland I	3·05
Midland II	2·93
East	2·66
South-West	3·00
Wales I	4·19
Wales II	4·65

The following list enumerates the counties grouped in Tables III and XII:

SOUTH-EAST
Bedfordshire
Berkshire
Buckinghamshire
Essex
Hertfordshire
Kent
London
Middlesex
Oxfordshire
Southampton
Surrey
Sussex, East
Sussex, West
Wight, Isle of

NORTH I
Durham
Northumberland

NORTH II
Cumberland
Westmorland
Yorkshire, E. Riding
Yorkshire, N. Riding

NORTH III
Yorkshire, W. Riding
York (County Borough)

NORTH IV
Cheshire
Lancashire

WALES I
Brecknockshire
Carmarthenshire
Glamorganshire
Monmouthshire

MIDLAND I
Gloucestershire
Herefordshire
Shropshire
Staffordshire
Warwickshire
Worcestershire

MIDLAND II
Derbyshire
Leicestershire
Northamptonshire
Nottinghamshire
Peterborough, Soke of

EAST
Cambridgeshire
Ely, Isle of
Huntingdonshire
Lincolnshire—
Parts of Holland
Parts of Kesteven
Parts of Lindsey
Norfolk
Rutlandshire
Suffolk, East
Suffolk, West

SOUTH-WEST
Cornwall
Devonshire
Dorsetshire
Somersetshire
Wiltshire

WALES II
Anglesey
Caernarvonshire
Cardiganshire
Denbighshire
Flintshire
Merionethshire
Montgomeryshire
Pembrokeshire
Radnorshire

The lowest rate was 2·48 in Greater London, and the highest was 4·65 in Wales II (North Wales). The large geographical differences in Table III and the suggested increment in the mortality in recent years (Table II) would seemingly indicate that injury at birth is a problem meriting attention. The number of deaths ascribed to this specific cause is not unimportant, as in 1945 in England and Wales the deaths of 1,785 babies under four weeks were specifically ascribed to injury at birth. Furthermore, on page 33 of report No. 94* on *Neo-Natal Mortality and Morbidity* it is stated: " Most observers agree that one of the leading causes of death in the first four weeks of life is injury at birth." For this reason it seemed desirable to study the available statistics in the hope that some light might be shed on this aspect of the mortality of infancy. Such is the purpose of the present investigation.

VARIATION IN RATES OF MORTALITY

In 1940 the Registrar General began the tabulation of deaths from injury at birth under one year in each administrative county and in each county borough

* Report by a joint committee of the Royal College of Obstetricians and Gynaecologists and the British Paediatric Association (1949). His Majesty's Stationery Office, London.

TABLE IV

MEAN DEATH RATE FROM INJURY AT BIRTH UNDER ONE YEAR

Area	Mean death rate under 1 year per 1,000 births	Coefficient of variation
Administrative counties ..	2·57	21
County boroughs	2·63	30

in England and Wales. Accordingly, it is possible to obtain a picture of the variation in the mortality throughout the country. The mean rate based on the statistical experience of 1940-43 with the inclusion of 1945 (the report for 1944 is not yet published) in areas with at least 5,000 live births during these five years and the relative percentage dispersion (coefficient of variation) around the mean rate (unweighted) were as in Table IV.

The death rate in the large towns is slightly greater than that in the administrative areas, and in both there is an appreciable dispersion in the mortality, the scatter (coefficient of variation) being relatively greater in the former than in the latter. The actual range in the mortality in the county boroughs was from 1·35 to 5·74 per 1,000 births and in the counties was from 1·47 to 3·98. Examples of high and low death rates per 1,000 live births are set out in Table V.

TABLE V

MEAN DEATH RATE FOR INJURY AT BIRTH

County boroughs				Administrative counties			
High		Low		High		Low	
Huddersfield ..	5·74	Merthyr ..	1·98	Anglesey ..	5·23*	Kent ..	1·99
Blackpool ..	5·03	Barrow ..	1·66	Brecknock ..	3·98	Notts. ..	1·79
South Shields ..	4·51	East Ham ..	1·62	Caernarvon ..	3·83	Berks. ..	1·76
Burnley ..	3·90	West Ham ..	1·35	Oxfordshire ..	3·26	Flint ..	1·47

* Rate based on less than 5,000 births.

The towns reveal a striking contrast. East Ham and West Ham, overcrowded East London boroughs, are at the bottom of the list, and Huddersfield and Blackpool, two towns differing considerably in their social and occupational environment, are at the top. In the administrative counties an equally striking comparison is evident. Oxfordshire, which invariably has one of the lowest rates from infant mortality, has a rate from injury at birth nearly double that of the contiguous county of Berkshire. The contrast between the county boroughs in these two counties (Reading and Oxford) is revealing (Table VI).

TABLE VI

DEATH RATE UNDER ONE YEAR FROM INJURY AT BIRTH AND NEONATAL DEATH RATE OF OXFORD AND READING

Area	Death rate under 1 year from injury at birth per 1,000 live births	Neo-natal death rate* (excluding injury at birth) per 1,000 live births
Oxford	2·68	15·0
Reading	2·18	23·2

* As it was not possible to obtain the death rate from injury at birth for the age period 0-4 weeks for each administrative county, each county borough and each metropolitan borough, the rate from injury at birth at 0-12 months has been subtracted from the total neonatal mortality. This is a legitimate step, since over 95 per cent. of the total deaths from injury at birth in England and Wales occur during the first four weeks of life. Hence it will be understood that in this paper the residual neonatal mortality equals the neonatal death rate from all causes minus the death rate from injury at birth at 0-12 months.

The death rate from injury at birth in Oxford is 23 per cent. in excess of that in Reading, whereas the neonatal rate is 35 per cent. in defect. The explanation of the divergence may be that in Oxford there is more accurate reporting, due to the teaching and team work of the obstetrical unit at the Radcliffe Infirmary; or that the social structure of the population of Oxford is, on the average, better than that of Reading and, as will be shown later, the mortality from injury at birth is correlated with social class, the death rate being greater among the higher than among the lower social groups of the population in England and Wales.

GEOGRAPHICAL DISTRIBUTION

In view of the disparity revealed by the foregoing comparisons, it was of interest to obtain a picture of the statistical importance of the mortality in the geographical counties of England and Wales, the geographical county being the administrative county including its county borough. For this purpose it

was deemed more satisfactory to compare the actual and calculated deaths, the " expected " or " calculated " deaths being deduced from the average statistical experience in the aggregated administrative counties and in the aggregated county boroughs respectively. An example will elucidate the procedure.

In the aggregated administrative counties of England and Wales the weighted average death rate under one year from injury at birth during the five years 1940-43 with the addition of 1945,* was 2·50 per 1,000 live births and in the aggregated county boroughs 2·75. Hence in the administrative county of Warwick, in which there were 38,508 live births during the period, the number of " expected " deaths from injury at birth would be

$$\frac{38{,}508 \times 2{\cdot}50}{1{,}000} = 96$$

Similarly in the county borough of Warwick the expected number would be

$$\frac{112{,}495 \times 2{\cdot}75}{1{,}000} = 309$$

Hence the calculated number of deaths from injury at birth in the geographical county would be

$$96 + 309 = 405$$

Finally the statistical significance of the difference between the actual and calculated number of expected deaths in each geographical county was estimated from the formula

$$\frac{\text{Actual deaths—Expected deaths}}{\sqrt{\text{Expected deaths}}}$$

and the results are depicted in Fig. 1 in which the shadings indicate counties with mortality significantly in excess, moderately in excess, moderately in defect, and significantly in defect. An example may help to facilitate its interpretation.

The difference in Warwickshire is positive, and, since it exceeds twice its standard error, it indicates an excessive number of deaths in this county. On similar reasoning the mortality in Berkshire is significantly in defect of what would be expected from the general average.

There is no definite belt or region of high mortality. The " black spots " are widely separated: Suffolk and Warwickshire in England; Pembroke, Caernarvon, Anglesey, and Denbigh in Wales. It is surprising to discover in Lancashire, where the infant mortality is invariably excessive, that the number of deaths from injury at birth is subnormal. The general picture in Fig. 1 certainly lacks the definition presented by Fig. 2, which relates to the disposition of the neonatal deaths excluding injury at birth. Here it will be observed that there are two distinct zones of excess and defect and the line of demarcation is from the Humber basin to that of the Severn. To the east of this line, including the counties of eastern and southern England generally, the mortality is low, whereas to the west and north the death rate is excessive. In view of this apparent dissimilarity in the geographical alignment of the two variables—injury at birth and the residual neonatal mortality—it was of interest to obtain a picture of the geographical distribution of the maternal mortality (Fig. 3). In it there are three black areas: one represented by Northumberland, Durham, and the North Riding of Yorkshire; the second, by Pembroke and Glamorgan; and the third, by Devonshire. In other respects the main features of this map are not unlike those outlined in Fig. 2.

A pictorial presentation based on the geographical county has an attraction, since it enables a quick impression to be gained, yet if one desires to make a precise comparison, the county as a unit may possibly be too large and so help to depress aspects inherent in the statistics of the large towns. For this reason it was resolved to evaluate the correlation coefficients between the three variables (1) injury at birth, (2) neonatal mortality (excluding injury at birth), and (3) maternal mortality in those county boroughs and administrative counties which had more than 5,000 births during the five years. Partial correlation coefficients were also calculated.

* The figures for 1944 are not yet available.

TABLE VII

DEATHS FROM INJURY AT BIRTH IN WARWICKSHIRE AND BERKSHIRE

	Actual No.	Expected No.	Difference	Standard error of difference
Warwickshire ..	466	405	61	3·03
Berkshire	57	78	—21	—2·38

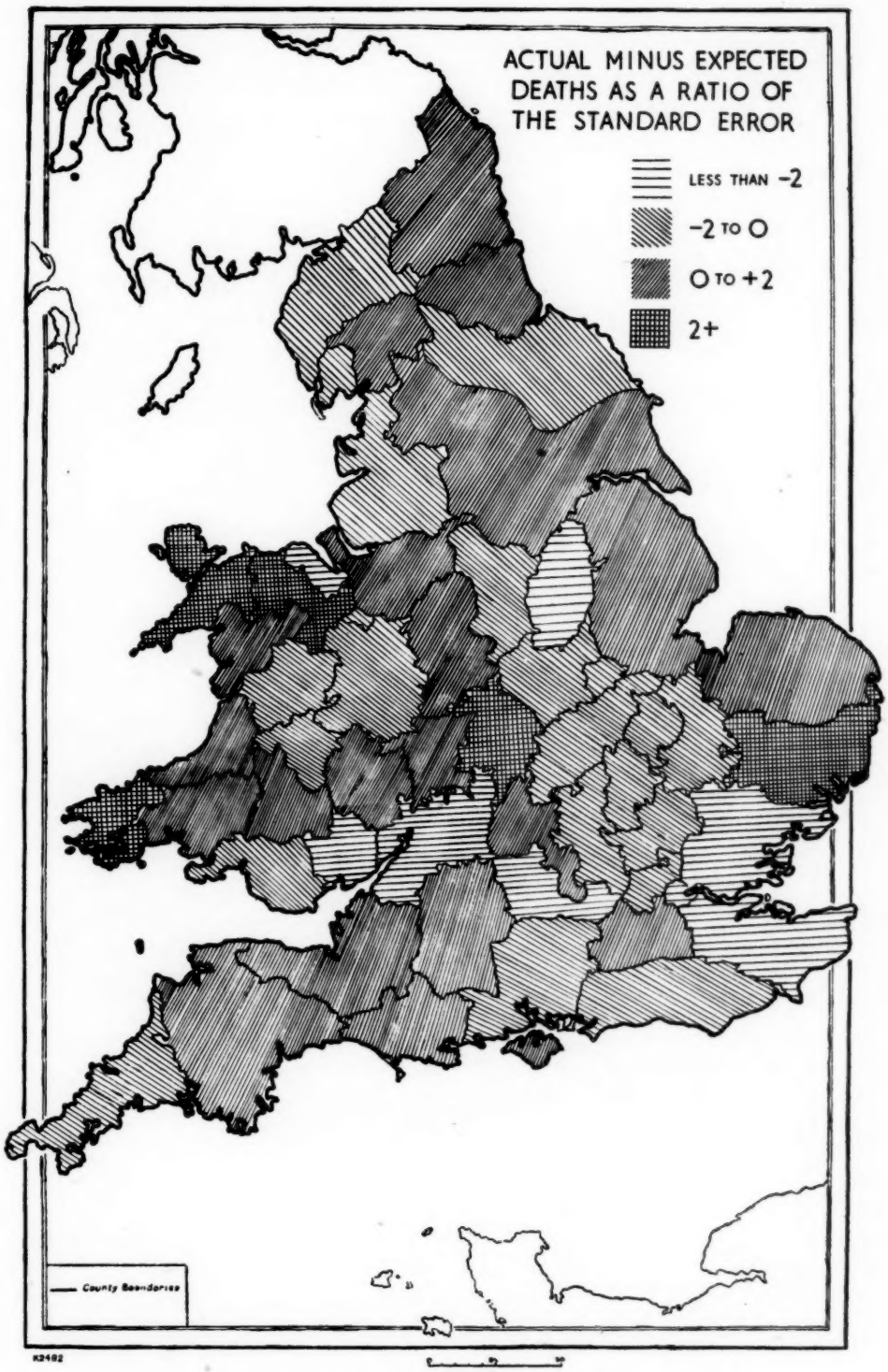

FIG. 1.—Map showing geographical incidence of injury at birth.

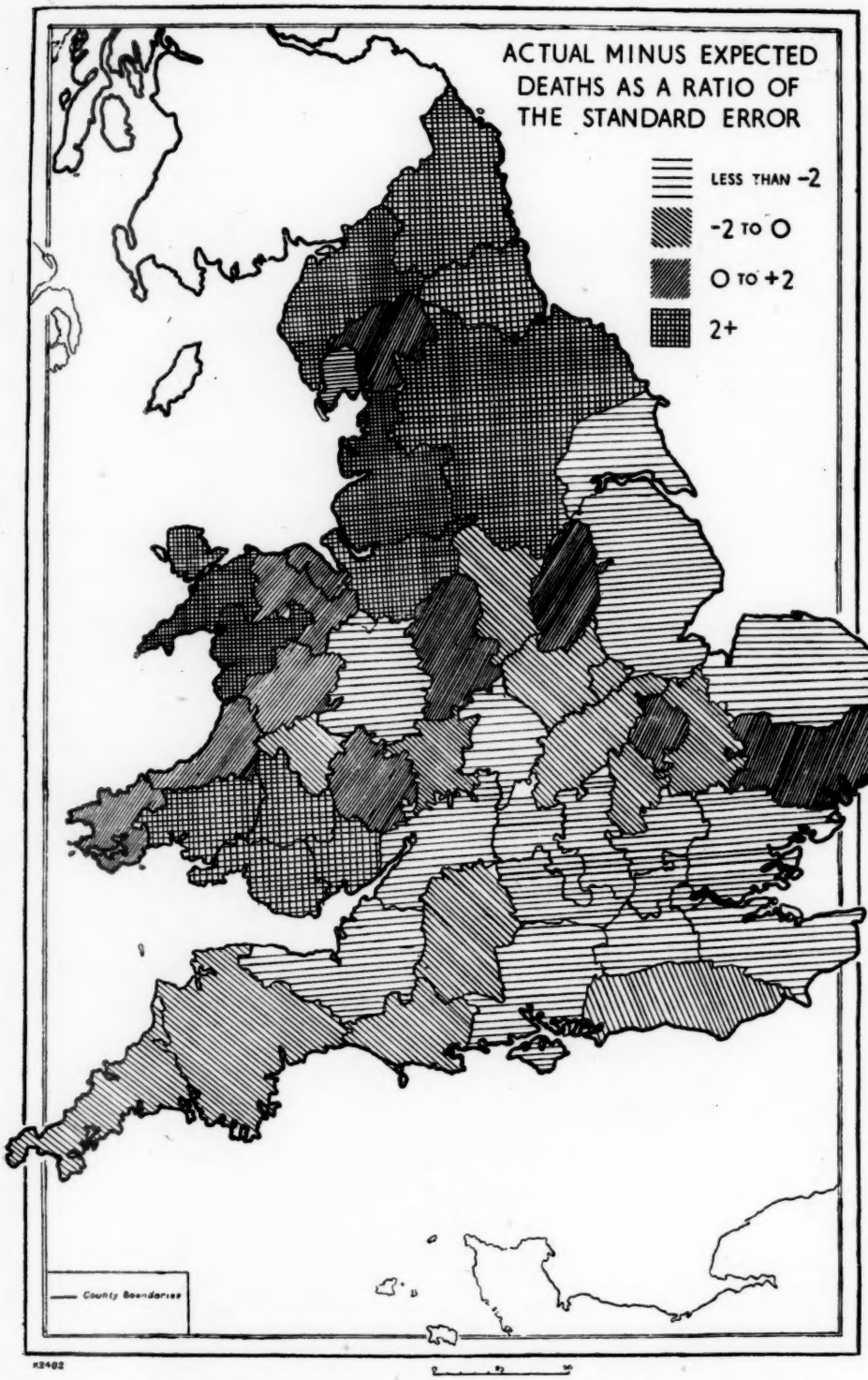

FIG. 2.—Map showing disposition of neonatal deaths excluding injury at birth.

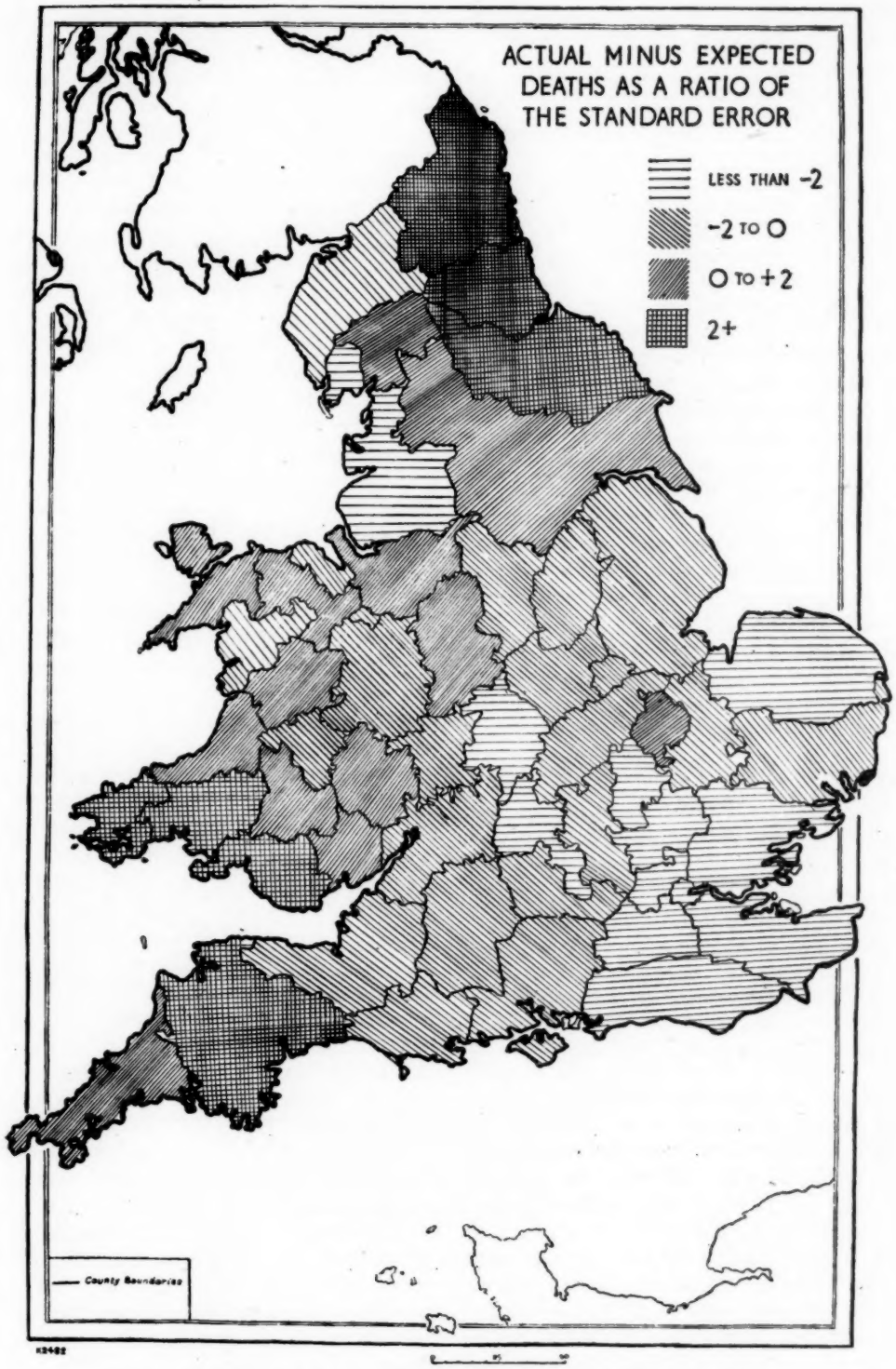

FIG. 3.—Map showing geographical distribution of maternal deaths.

These measure the correlation between two of these variables when the effect of the third is eliminated. The results were as follows:—

Administrative counties (52)

r12 = + ·072	r12·3 = — ·014
r13 = + ·136	r13·2 = + ·166
r23 = + ·614*	r23·1 = + ·621*

County boroughs (72)

r12 = + ·233†	r12·3 = + ·193
r13 = + ·143	r13·2 = + ·057
r23 = + ·399*	r23·1 = + ·379*

* Statistically significant. † Borderline significance.

It will be noted that there is only a very small correlation between injury at birth and either the neonatal or maternal mortality. In other words, the factors which play a part in determining the mortality from injury at birth are relatively unimportant so far as the residual neonatal (dominated, of course, by prematurity) and maternal mortalities are concerned. On the other hand the correlation between the neonatal mortality and maternal mortality is substantial, particularly in the administrative counties, where the partial coefficient is + ·621. Hence one can safely conclude that towns or administrative counties which have a high death rate from injury at birth do not necessarily have a proportionately high neonatal mortality.

London

Since more births occur in hospital in London than in other parts of the country, a separate analysis was made for the 28 metropolitan boroughs. The correlation between the three variables was as follows:—

r12 = + ·266	r12·3 = — ·033
r13 = + ·482*	r13·2 = + ·438†
r23 = + ·520*	r23·1 = + ·481*

* Statistically significant. † Borderline significance.

The results for the London boroughs confirm those in the county generally in as much as the partial coefficients indicate the dissociation between the mortality from injury at birth and the neonatal death rate; and the positive relationship between neonatal and maternal mortality.

They differ, however, in one aspect, because there is evidence that there is a fairly definite relationship between injury at birth and maternal mortality, the partial coefficient being + ·438. What is the explanation of this ? Can it be that there is more surgical interference in deliveries in hospital, or is it simply that a high proportion of difficult cases are delivered in hospitals ?

Possible Causes

In view of the suggested increase in the mortality from injury at birth in England and Wales generally since 1921, it is cogent to consider the possible causes under three headings.

An Increasing Proportion of First Births.—These are generally accepted as being more difficult than subsequent deliveries. If this is so there should be a positive correlation between the rate from injury at birth in an area and the proportion of first births there. It is possible to obtain a crude answer on this aspect because the Registrar General has published data showing the proportion of first births in the 12 major divisions of England and Wales. Correlating these ratios for the period 1940-42 with the corresponding death rates from injury at birth, the coefficient was found to be r = – 0·201, a result which is statistically insignificant.

Significance of Hospital Deliveries.—In London, approximately 80 per cent. of the total births occur in institutions, as compared with approximately 20 per cent. in the remainder of the country. The corresponding rates of mortality from injury at birth are 2·48 and 2·56 per 1,000 births respectively. In view of the disparity in the proportion of births which occur in hospitals in London compared with the rest of the country and the approximate equality in the rates of mortality (assuming that the data are comparable in all other respects) it would seem that birth in hospital is an ineffective factor in preventing death from injury at birth. It may, of course, as has been suggested, be argued that the more difficult cases are now usually removed to hospital for delivery. It is perfectly true that difficult cases are referred to doctors by midwives but, as regards delivery in hospital in London the greater part of the enrolment is made early in pregnancy before a prognosis is possible, excepting in so far as anatomical abnormalities in the mother are recognizable in a proportion of cases.

Instrumental Delivery.—There has been an increasing tendency in recent years to have recourse to instrumental delivery. There are no specific statistics to support this statement, but the contrast of the mortality rates according to social status may act as a pointer.

According to the statistics compiled for the period 1930-32 by the Registrar General, the death rate from injury at birth and the residual neonatal mortality in three broad social groups in England and Wales in the period 1930-32 was as set out in Table VIII.

It will be noticed that with descent in the social scale the mortality from injury declines, whereas for the residual neonatal mortality there is an

TABLE VIII

COMPARISON OF DEATH RATE FROM INJURY AT BIRTH AND RESIDUAL NEONATAL MORTALITY IN THREE SOCIAL GROUPS

Classes	Legitimate births	
	Injury at birth under 1 year per 1,000 live births	Neo-natal mortality (excluding injury at birth) per 1,000 births
I and II (professional and administrative grades) ..	2·44	23·9
III (skilled workers)	2·13	27·3
IV and V (semi-skilled and unskilled labourers)	1·96	30·3

increase. Obviously different agencies operate to produce this divergency.

Further evidence of the pattern made by social class is revealed by the statistics of a more recent date. When the boroughs of London are classified into two broad groups according to socio-economic status "good" and "poor", and the expected number of deaths is calculated for the quinquennial period 1940-43 (including 1945) and compared with those actually enumerated, the following results are obtained (Table IX).

TABLE IX

ACTUAL AND EXPECTED DEATHS FROM INJURY AT BIRTH IN LONDON BOROUGHS GROUPED ACCORDING TO SOCIO-ECONOMIC STATUS

District	Injury at birth		Difference
	Actual	Expected	Standard error of difference
Good ..	319	306	+0·74
Poor ..	204	221	−1·15

It will be seen that there is a tendency for the "good districts" to have a mortality in excess of the average and for the "poor" areas to be in defect, although in neither instance is the difference statistically significant. The picture, while it is suggestive, is not as definite as is that for maternal mortality in the two groups (Table X).

In the "good" districts the recorded maternal deaths are significantly in excess of the number expected to occur, whereas in the poor areas they are statistically below expectation. It may be argued that the correlation which has been indicated

TABLE X

ACTUAL AND EXPECTED MATERNAL DEATHS IN LONDON BOROUGHS GROUPED ACCORDING TO SOCIO-ECONOMIC STATUS

District	Actual	Expected	Difference
			Standard error of difference
Good ..	295	261	+2·10
Poor ..	158	192	−2·45

may possibly be due to a higher proportion of first births in the better class districts but this argument invites the question, Is injury at birth relatively more frequent amongst first births ? The available statistical evidence based on the Registrar General's returns, to which reference has already been made, offers no confirmation of this idea. The correlation coefficient between injury at birth and the proportion of first births was statistically insignificant.

AGE OF MOTHER

Since age is an important correlate of mortality statistics it may be asked to what extent the age of the mother is a dominant factor in the present instance. Is injury to the baby at birth relatively more frequent among older than younger mothers, particularly if they are primiparous women ? The only available evidence on this aspect of the problem on a national scale was that relating to stillbirths in Scotland during the years 1939-45. The death rate ascribed to injury at birth among the babies in this series of observations when correlated with the age of the mother was found to be as recorded in Table XI.

TABLE XI

DEATH RATE IN SCOTLAND FROM INJURY AT BIRTH IN STILLBIRTHS CLASSIFIED ACCORDING TO AGE OF MOTHER

Age of mother	Still-births per 100,000 (live and stillbirths)
Under 35 years	90
Over 35 years	112

It would appear from these values that the age of the mother is a factor of importance. This association invites the question, Has there been an increase in the age of motherhood ? Unfortunately the relevant statistics are only available for a restricted period, because it was not until 1939 that the statistics of births in relation to the age of mothers were first published in England and Wales. Comparing the mean age of legitimate maternities for first children in 1939 and in 1945 the values in the 12 major divisions were as in Table XII.

TABLE XII

MEAN AGE OF LEGITIMATE MATERNITIES IN TWELVE MAJOR DIVISIONS OF ENGLAND AND WALES

Area*	Mean age of legitimate maternities (first children)		Difference
	1939	1945	
Greater London ..	26·8	26·9	+0·1
Remainder of South-Eastern Counties	26·4	26·5	+0·1
North I	25·6	25·8	+0·2
North II	25·9	26·1	+0·2
North III	26·2	26·3	+0·1
North IV	26·6	26·7	+0·1
Midland I	26·3	26·5	+0·2
Midland II ..	26·3	26·2	−0·1
East	26·0	26·0	—
South-West ..	26·5	26·4	−0·1
Wales I	25·6	25·8	+0·2
Wales II	26·7	26·9	+0·2

* See Table III for details of areas thus grouped.

It will be seen that in the majority of instances there has been an increase in the mean age, but it is not of sufficient moment to explain the ostensible increase in the mortality from injury at birth. Furthermore, the mean age of mothers in Greater London is almost identical with that of Wales II (North Wales). There is, however, a wide difference in the mortality rates from injury at birth, as is indicated by the composite figures presented in Table III.

SUMMARY AND CONCLUSIONS

In this study we have considered only fatal injuries at birth, as no data are available in connexion with the numerous residual disabilities resulting from non-fatal injury. This analysis of the available statistics in England and Wales since 1921, and in various administrative counties and county boroughs including London, may be summarized as follows.

There has been an apparent increase in the mortality during the period 1921-45 even when allowance is made for the transference of deaths from convulsions, asphyxia, and atelectasis.

There are large differences between the mortality rates in the various administrative counties, and likewise between the county boroughs.

Contrary to expectation, there is little correlation between the mortality from injury at birth and the proportion of first births in the 12 major regions of England and Wales.

There is evidence from Scottish data on still-births that the mortality ascribed to injury at birth among them depended upon the age of the mother, the death rate being higher amongst older women. There are no available statistics in England and Wales which relate the age of the mother to the mortality from injury at birth. Statistics of the age of maternity have been published since 1939 and have indicated a slight increase in the mean age between 1939 and 1945 throughout the country. It is very doubtful, however, if this increase is sufficient to account for the suggested increase in the mortality.

The mortality in a group of " poor " boroughs in London is subnormal, whereas in a " residential " group it is in excess of the average for London. There is also a positive correlation between the mortality from injury at birth and the maternal death rate. This may or may not be the result of undue surgical interference.

The evidence presented would seem to indicate the reality of the problem, but complete proof of its existence, and, if it does exist, of its solution, can be determined only by a carefully planned specific study.

The need here demonstrated for such a study has recently been emphasized in the following statement from the Report on Neonatal Mortality and Morbidity.

" Many cases of birth injury are undoubtedly due to lack of obstetrical skill and judgement on the part of the accoucheur, especially when delivery by breech or by forceps has been necessary."

ABSTRACTS

(This section of the JOURNAL *is devoted to selected abstracts of articles on social medicine appearing in the current literature. The section will be edited in collaboration with the two abstracting Journals,* Abstracts of World Medicine, *and* Abstracts of Surgery, Obstetrics and Gynaecology.)

A Genetical Study of Human Mammary Cancer. PENROSE, L. S., MACKENZIE, H. J., and KARN, M. N. (1948). *Ann. Eugen., Camb.*, **14**, 234.

The role of hereditary factors in the aetiology of cancer is most easily recognized in the case of mammary cancer because the latter is more accurately diagnosed than malignant growths at most other sites. Furthermore, in the great majority of instances such tumours produce a clearly defined range of pathological conditions.

The history of investigations into the genetic aspects of mammary cancer was summarized by Jacobsen in 1946. In spite of the evidence brought forward by a great many investigators that mammary cancer frequently affects several related females in a pedigree doubt still remains whether hereditary factors play any significant part in its causation. This uncertainty derives from the fact that this particular disease is very prevalent in the population as a whole, being responsible for nearly 3% of all deaths in females. It is thus difficult to demonstrate that any familial concentration is not merely the result of a random distribution of cases.

In the present study a direct comparison is made of deaths from different types of cancer in the relatives of a series of propositae with the rates for the population as a whole. The material was compiled in such a way that if the results did show definite evidence of hereditary transmission indications of the type of inheritance could also be obtained. Particular attention was paid to the possibility of transmission through maternal milk. All patients suffering from mammary cancer and attending a clinic were personally interviewed and family histories including those of patients' parents, brothers, sisters, children, grandparents, uncles, and aunts were compiled, 510 cases of mammary cancer in females being collected. In 144 instances a relative suffered from cancer of some type and in 118 instances one or more relatives had mammary cancer. The mean age of the patients when first interviewed was 55·2 years and they had on the average lived 3½ years since the first signs of the disease had been recognized; 255 were affected first on the left side and 253 on the right side. In the rest there was some uncertainty on this point. Of the mothers of these propositae 25 had died of mammary cancer and 51 other mothers had died of some other form of malignant disease. At the time of the investigation 104 mothers were still living and of these 6 were known to be suffering from mammary cancer. No similarity between age of onset of breast cancer in the mothers and age of onset in the propositae could be demonstrated. Some 365 sisters of the propositae had died, 23 of mammary cancer and 19 of other types of malignant disease.

There was thus a highly significant increase in observed deaths from mammary cancer amongst these sisters but no significant divergence from expectation as regards deaths from other types of malignant disease. Amongst 890 sisters still living at the time of the investigation 24 were under treatment for breast cancer and 7 for other types of malignant disease. Only one death due to mammary cancer was found amongst the male relatives. Amongst the propositae were 410 married women who had had 387 liveborn sons and 398 liveborn daughters together with 28 stillbirths. The group of propositae was therefore slightly less fertile than the general female population from which it was drawn. The daughter of one proposita was found to be suffering from mammary cancer and 2 sons of 2 others had died of malignant disease.

There emerged from this study, therefore, a strong suggestion that transmission of a specific genetical factor is a major cause of mammary cancer. The familial incidence amongst sibs, however, was not sufficiently high to suggest any simple Mendelian explanation of the inheritance. The search for evidence that mammary cancer in the human female was being inherited through maternal milk gave equivocal results, no significant association being found between patients who were breast-fed in infancy and those in whose families there was concentration of the disease in the maternal relatives.

F. A. E. Crew

A Statistical Report on 2,529 Cases of Cancer of the Breast. HARNETT, W. L. (1948). *Brit. J. Cancer*, **2**, 212.

A statistical study is made of 2,152 primary and 377 recurrent carcinomata originating in the breast. The civil state and age distributions are first shown and compared with those in Lane-Claypon's series. As the author's data include recurrences, the mean ages are higher than those in Lane-Claypon's report which dealt with patients coming to hospital for operation. It appears that the interval between recognition of first symptoms and consultation of a doctor was more than 6 months in 40·6% of the patients. Rather fewer than a quarter of the patients, 24·3%, had growths in clinical stage I, namely, growth confined to breast, no involvement of axillary lymph nodes, and no infiltration of skin or muscles. Of patients with stage-I growths treated by radical mastectomy (236) 68·2% of those traced survived 5 years. Of those treated by radical mastectomy combined with radiotherapy (69) the percentage was 64·1. Detailed tables, too numerous for abstraction, bring into account age, duration of symptoms, and other factors. There is an elaborate and valuable tabulation of survivorship in which are shown the mean actual survivorship, the expected survivorship (from English Life Table No. 10), and the maximum possible for 5 years (60 months). This is undoubtedly the right method of analysis. The conclusion emerges that the 5-year expectation of life after radical mastectomy ranges from 91·03% of normal in stage I down to 68·4% in stage IIIb (growth infiltrating skin or muscle or both, and axillary lymph nodes involved).

(It is to be hoped that a similarly detailed analysis will be made of data covering a further range of years;

inevitably, sub-tabulation of even so long a series as this ends in small numbers the statistical interpretation of which must be difficult. The paper is, however, an important one.) *Major Greenwood*

Childbearing in the Twilight of the Reproductive Period. DAVIS, M. E., and SESKI, A. (1948). *Surg. Gynec. Obstet.*, **87**, 145.

The authors point out that the function of childbirth is a function of youth and not of middle age. They submit a statistical survey of childbearing in all women of 40 years or over confined in the Chicago Lying-in Hospital during 1927-1944. This comprises 1,011 patients out of a total of 52,128 (1·94%); 16·8% of the 1,011 were primigravidae. Among the parous patients, an interval of 10 years or more had elapsed since the previous child in 18%. The incidences of various complications of pregnancy were studied in the 1,011 patients and compared with the general figures for the hospital. Abortion, placenta praevia, and toxaemia of pregnancy were all commoner. The actual figure for the last-named was 24%, compared with 7·1%. A higher proportion than usual was delivered by Caesarean section (13·3% against 4·4%). This difference is partially accounted for by a rate of 31·5% in elderly primigravidae, although the rate in multiparae (9·5%) was also above the usual figure. The Caesarean section rates for dystocia and disproportion did not differ materially in the two series, the increase in abdominal delivery being due chiefly to the increase in pregnancy toxaemia and ante-partum haemorrhage in the older group. In those delivered vaginally, the proportions in which delivery was completed by the natural forces were approximately equal, but among forceps deliveries there were relatively more mid-forceps deliveries than low forceps in the elderly patients. There was a striking increase in breech presentation (7·4% in primigravidae and 5·4% in multiparae, against 4·4% for the hospital). The weights of the children showed a wider scatter in the older patients, with an increase in the numbers of very large children and also of premature infants. Six mongols were recognized whilst in hospital. Foetal deformities were present in 2·7%, against 1%. The total foetal mortality rate was 9·5% compared with 3·5%. Four mothers died, all before 1931. By present day standards, the authors consider that three of these deaths should have been avoided.

They conclude that their figures indicate a definite increase in the hazards for mother and for child, but emphasize that these figures do not reveal the whole story, and that they do not include evidence of the recovery of the women and their babies after delivery. Their impression is that the middle-aged mothers recover more slowly and are more likely to have persistent minor complaints, but, on the other hand, they have been impressed with the rejuvenating effect of motherhood upon some of their patients. They have not been able to evaluate the remote prognosis for the mother who develops serious complications of pregnancy and labour. *W. I. C. Morris*

Abortion as a Factor in Sterility (L'aborto quale fattore di sterilita). NOBILE, T. (1948). *Ginecologia, Torino*, **14**, 528.

Study of the case records of 2,000 women treated in the Sterility Clinic at Turin during the years 1939-1944 leads to the conclusion that abortion is an important factor in sterility; 204 women were sterile after abortion and of these 75% had had only one abortion. Premarital abortion was verified in 14%; 12% had had a febrile abortion. The period of infertility after the abortion varied from 1 year to 15 years, but 25% of patients attended the clinic within the first 2 years after abortion. Clinical signs of tubal infection were present in 23·5%. Out of 60 women examined radiologically there was evidence in 52% of tubal occlusion not suspected on clinical examination. Evidence that even a single abortion can cause sterility is given by the fact that 132 out of the 204 patients were aged from 25 to 35, normally the period of maximum female fertility. Apart from subsequent infection, abortion causes serious disturbance of function, while psychological factors are also probably of importance. *Josephine Barnes*

Observations on the Problem of Perinatal Mortality (Příspěvek k otázce perinatální úmrtnosti). VÁCHA, K. (1948). *Čsl. Gynaek.*, **13**, 264.

The author discusses the causes of stillbirth and of neonatal mortality in the Obstetrical Department at Ústi, Czechoslovakia, in 1946 and 1947. The causes of infant death are divided into 15 groups. Debility in the premature infant was responsible for the largest number. In 1947 prematurity accounted for 73% of the stillbirth and neonatal death rate. If we deduct these infants we get the relative death rate of 1·8%. From this the author comes to the conclusion that the infant death rate does not depend so much on the conduct of labour in obstetrical institutions, but that it could be considerably lowered by improving antenatal care.—(Author's summary.)

Further Observations on the Medico-Social Aspect of Venereal Diseases. MACFARLANE, W. V. (1948). *Publ. Hlth, Lond.*, **62**, 4.

The Venereal Diseases Department at the Newcastle General Hospital is computed to serve a population of approximately 1,000,000. New cases of syphilis seen at the clinic totalled 798 in 1927, 350 in 1932, 390 in 1937, 720 in 1942, and 583 in 1947. The number of patients suffering from gonorrhoea who attended in the same years was 1,070, 859, 932, 874, and 1,033, respectively. The author stresses what he calls the "medico-social unit" which, in addition to the chief and two assistant almoners and clerical staff working in the clinic, has 3 full-time and 3 part-time health visitors to undertake domiciliary visits.

Of 2,855 patients interrogated by the unit only 58% gave reliable information concerning the contact, but 1,560 contacts were examined and 70% were found to have venereal disease. The meeting places in the case of 1,000 contacts interviewed about the source of casual infection were public house (652), the streets (177), railway stations (95), dance halls (67), private houses, cinemas, cafés, coffee stalls, ships and docks, brothels, theatres, hostels, and public parks. In 32% of cases intercourse took place in the house of one or other partner and in 16% in brothels.

The case records of 43 promiscuous women showed that 52% had previously been convicted in the courts and that 73% had intelligent quotients below normal. At the time of examination 16 were working in factories, 8 were in domestic service, 5 were waitresses, 4 were barmaids, 2 were shop assistants, and 8 did not work at all.

"Case-holding" is achieved by a combination of

personal letter and home visit; the latter has been found to be very successful, the ratio between successful and unsuccessful visits being 1 : 1. It is, however, considered that if there is no response after two visits and two letters little further can generally be achieved. The corrected defaulter rates for male and female patients with syphilis and gonorrhoea vary from 6 to 13%. Of 3,478 women who had defaulted for more than 2 years 2,032 were brought back to the clinic as a result of these measures; of male defaulters no less than 70% returned to the clinic after one or two letters only, but 36% were regarded as completely irresponsible. The introduction of penicillin with its more rapid apparent cure, has led to a marked increase in default; 54% of patients default in the month immediately after treatment.

(This clinic is one of the very few in Britain which publishes its statistics, and these are therefore read with considerable interest. It is idle to suggest that the continued high rate of new cases is no advertisement for the preventive measures of the area, because it can always be claimed that this is the result of better case-finding. However, more attention might be paid to case-finding than to case-holding, which, in these days of penicillin therapy, can no longer be said to be a problem of public health proportions even if it is a pressing one for a few unfortunate individuals.)

R. R. Willcox

The Health of Towns Association in Great Britain 1844-1849. An Exposition of the Primary Voluntary Health Society in the Anglo-Saxon Public Health Movement. PATERSON, R. G. (1948). *Bull. Hist. Med.*, **22**, 373.

At the present time, when health legislation is so widely discussed, it is interesting to recall the pioneer effort of the " Health of Towns Association " a century ago. Founded by Dr. Southwood Smith on December 11, 1844, the movement may be considered as the prototype of the many present-day health organizations in Great Britain and the United States. This essay in health reform was one of many such efforts made in various fields about this time.

At the end of the eighteenth century a virulent outbreak of typhus fever among the mill-workers of Manchester led to an investigation of their conditions and to the inception of various means to improve them. Another powerful impetus to sanitary reform was the cholera epidemic of 1831-2. The close connexion between poverty and disease came into sharp focus. A further step in advance was the registration of births, deaths, and marriages, which became effective in 1837.

In 1838 Edwin Chadwick persuaded the Poor Law Commissioners to inquire into the physical causes of sickness and mortality, and the consequent reports acted as a stimulus to further effort. Dr. Southwood Smith, who had been an active participant in some of the earlier reforms, decided to take measures to influence the public and Parliament in the direction of health legislation. The new association, which held its first meeting at Exeter Hall, London, was not a charity, but aimed at preventing the evils which charities endeavoured to palliate. " It would substitute health for disease, cleanliness for filth, economy for waste, justice for charity." Provincial branches were soon formed. One of the most active was that of Liverpool, which published a monthly periodical, *The Liverpool Health of Towns Advocate*, which appeared from 1845 to 1847. Support to the movement was also given by those who were at that time known as the working classes, and thus came into being, in London, " The Metropolitan Working Classes Association for Improving the Public Health." Among the objects of this association was the diffusion of information concerning the need for cleanliness, ventilation, drainage, and the importance of the nursing, feeding, and clothing of children.

The work of the Health of Towns Association soon bore fruit, and in 1848 the Public Health Act was passed, embodying many of the ideals for which the association had striven. The General Board of Health came into being, and instituted inquiries into the prevalence of infectious diseases, the disposal of the dead, water supplies, and other essential matters which concerned the public health. The first President of the Board was Sir Benjamin Hall. With the passage of the Public Health Act of 1848 the need for the association was no longer felt, and its activities ceased. Nevertheless, the Health of Towns Association, during its brief career, began a process of health education which led to effective legislation. The fruitful results of its work illustrate the importance of voluntary and unofficial efforts as a means of preparing the ground for the improvement of the general health of the community by well-directed laws.

Douglas Guthrie

Socio-emotional Factors Accounting for Growth Failure in Children Living in an Institution. FRIED, R., and MAYER, M. F. (1948). *J. Pediat.*, **33**, 444.

The authors, working at Bellefaire, the Jewish Children's Home at Cleveland, Ohio, claim that by using the Wetzel grid technique (a specially designed growth chart) they have shown that socio-emotional factors tend to affect physical growth adversely. They also claim that psychiatric evidence and observations of behaviour, combined with medical findings on health, support their contention. They believe that the best conditions of shelter, food, schooling, and medical and social supervision are in themselves insufficient to ensure normal growth in the presence of psychological disturbance.

They claim that the grid technique, besides serving as a control chart on physical growth, has proved valuable in co-ordinating the work of the specialist workers—social workers, psychiatrists, paediatricians, nurses, and supervisors—at the home. Quarterly readings of height and weight are part of the routine supervision of every child. Failure to develop normally is taken as a signal for monthly observations, and also for a search for the cause, physical or psychological. Two types of growth failure—simple nutrition and obesity—are recognized. Children newly admitted show measurable growth failure (usually malnutrition) in 75 to 90% of cases. Four case records are included, illustrating: (1) prolonged initial adjustment; (2) delayed and intractable maladjustment; (3) recurrent failure; and (4) interrupted care, with recovery on a second admission.

(Although the numbers of cases in this paper are too small to be of statistical value, they serve to emphasize that children, being composed of psyche and soma, will, if chronically unhappy or unsettled, fail to develop physically as normally happy children would.)

A. T. Macqueen

The Physical and Mental Development of Premature Infants. A Statistical Survey with a Five-year Follow-up. HIRSCHL, D., LEVY, H., and LITVAK, A. M. (1948). *Arch. Pediat.*, **65**, 648.

Prematurity is still the chief cause of infant mortality. Infants admitted to a nursery for premature babies

between 1942 and 1946 were studied. Their weights at birth varied from 694 g. to 2,000 g. The mortality rate was 24·1%, and was higher for males than for females. The death rate varied inversely with the weight groups of the babies. The causes of death included cerebral haemorrhage, atelectasis, erythroblastosis foetalis, haemorrhagic disease of the newborn, intestinal obstruction, aspiration pneumonia, and bronchopneumonia. The authors point out that the physical and mental development of these children is slightly delayed but is ultimately normal—for example, the males reach a normal weight by the end of 3 years and the females by the end of one year and they are all slower in walking and talking than are full-term infants.

(In spite of recent advance in the care of premature infants the death rate has not decreased. The very small babies weighing 1,000 g. present the greatest problem. Abrahamson has grouped the causes of mortality into: diseases of the mother; duration of labour and obstetrical procedures; use of drugs in labour; disease of the baby; and after-care of baby. It is emphasized that the obstetrician and the pediatrician have each their part to play in bringing about a decrease in the mortality rate.)

B. S. P. Gurney

Growth of the Preschool Child in London. GORE, A. T., and PALMER, W. T. (1949). *Lancet*, **1**, 385.

This investigation was undertaken to supply the medical officers of clinics with data on weights and heights of children under school age, until a comprehensive survey could be made of the country generally. Measurements were taken in four London boroughs, and it is thought from evidence obtained of social conditions that samples representative of all London have been obtained. The survey included 2,881 boys and 2,803 girls and the measurements were taken from records kept by the four borough medical officers. In a table graduated mean weights are given and the authors state that data extracted from this table would be more useful for clinic work than a normal single curve of average weight. (At one year the weight for boys is slightly below that of a 1940-2 Glossop survey (23·13 lb.; 10·5 kg.) and slightly above that of the girls in the Glossop survey (20·56 lb.; 9·3 kg.).) Another table gives the actual averages of the measurements of height, but the authors consider the number measured (921) too small to be of any great value; however, as reliable height measurements are not readily available for very young children, the figures should prove useful. (Workers in clinics will find these tables valuable for reference purposes.)

E. H. M. Milligan

The Recent Trend of Diphtheria in England and Wales. MARTIN, W. J. (1948). *Mon. Bull. Min. Hlth*, **7**, 232.

Before the introduction of widespread immunization, mortality rates from diphtheria in England and Wales fell from 888 per 1,000,000 children under 15 in 1901 to 301 in 1930. In 1940 the Government offered a prophylactic free to all local authorities. Since then the mortality has decreased more rapidly than the incidence: in 1946 deaths were 15% and notifications 30% of the pre-war levels. As a cause of death at ages 1-5 diphtheria was the third highest in 1937-8 and sixth in 1945; at ages 5-10 it was first in 1937-8 and third in 1945. The decline in attack rate has not been uniform throughout the country; London has had the most rapid fall, and the northern, south-western, and west midland counties and Wales the least. Urban districts show a slightly greater decline than rural. There had been a change in the age distribution of notifications, and in 1947 the pre-school child and adult were relatively more often attacked than in 1944, whereas the attack rate for age group 10-15 was in 1947 one-fifth of that in 1944. This is in direct contrast with the experience of preceding decades.

A. Michael Critchley

Job Performance of Physically Impaired Persons in Industry. FELTON, J. S. (1948). *Occup. Med.*, **5**, 466.

The Oak Ridge National Laboratory employed 452 (11·1%) people with physical limitations. The performance of 300 of these is compared with that of 300 normal controls. The classification of applicants for work were as follows: (1) any work; (2) any work, in spite of minor defects; (3) certain jobs, with specified restrictions of activity; and (4) no employment. Those with limitations were "placed" after reference to analyses of job requirements; 59% fell into category (2) and 13% into category (3). Their performance was measured on the opinions of their supervisors, a separate rating being given for quality of work, quantity of work, knowledge of the job, adaptability, judgment, dependability, attitude, attendance, punctuality, and ability. In the hope of getting greater objectivity, supervisors were given a standard form to complete in which reasons for each rating were set out.

Each group comprised workers in many trades. The groups were fairly comparable as regards age, sex, race, marital state, and length of employment. The disabled men had all manner of abnormalities: 32·6% had hernia. An assessment of emotional adjustment, based on the answers given to significant items in a questionnaire consisting of 200 queries (Cornell Index), was made for comparison. Accident-rates also were compared. The average scores of the two groups did not differ as regards punctuality, attendance, or attitude, but the quantity of work done appeared to be greater in the "impaired" group, despite the variety of jobs, while scores for ability and quality of work were rather better in the unimpaired. The impaired workers had a slightly higher average Cornell Index (that is, they had a greater tendency to neurosis). The physically normal group had a significantly higher accident rate, but the number of injuries per worker was the same in both groups. The total scores suggest no significant difference between the job performance of the normal and impaired groups, nor between the larger subgroups of the impaired, for example, those with cardiovascular disease, hernia, or tuberculosis. There was a low correlation between the Cornell Index scores and the supervisors' ratings; the former gives little indication whether or not a man is adjusted satisfactorily to his job. The author concludes that these performances reveal the fallacy of applying rigid physical standards in industry, and that disabled persons are valuable, productive, and safe employees. (Physical impairment and disability are confused here. The former was determined at pre-employment examination, and all varieties seem to have been included, regardless of whether a worker was handicapped in the job he was given. Measurements of job performance take no account of individual workers' problems. Many workers left before their performance could be studied.)

J. N. Agate

Some Notes on Suicide. O'Connor, W. A. (1948). *Brit. J. med. Psychol.*, **21**, 222.

An explanation of suicide is worked out on the basis of Freud's theory of the conflict between Life and Death, Eros and Thanatos. Eros is constantly active in maintaining life but there is at the same time a conflicting pull towards death and rest, and it is suggested that the longing for immortal life, which can only be reached by death, may belong to this side of the conflict. The frequently observed calmness and complacency of a patient just before he commits suicide may be due to a solution of the conflict in favour of Thanatos. Obviously church and state condemn suicide more fiercely than can be justified by the infinitesimal danger to the biological survival of the human race. This condemnation is quite disproportionate when compared with the indifference of those institutions to poverty and disease. The explanation of such official severity would seem to be the fear that individuals may too easily succumb to the demands of the death instinct, although this fear is inconsistent with the reverence for martyrs and monastic self-flagellators.

The death wish is certainly not conscious but is deeply buried in the unconscious, and so it is only when the ascendency of the conscious mind is disturbed in severe mental illness that suicide is likely to occur. In this state phantasy can become dominant and the wish may become a command. Suicide may become a certainty when the reality principle falls entirely into the background. Until this happens real suicide is unlikely. Reality worries are not usually responsible for suicide, and however devastating the viscissitudes of life may be it is doubtful if they should ever be looked upon as liminal stimuli for actual self-destruction. The author quotes 18 cases which came under his own observation and considers that 90% of suicides had led an unsatisfactory sexual life and that 50% had homosexual tendencies.

R. G. Gordon

GENERAL ADVISORY BOARD

The Editors of the *British Journal of Social Medicine* are anxious to keep in close touch with centres of social medicine in other parts of the world. To this end they are forming a General Advisory Board. The Editors note with pleasure that the following have already expressed their willingness to serve :

Dr. C. J. Brenkman (Joint Editor of the *Tijdschrift voor Sociale Geneeskunde*).

Dr. L. Derobert (*Archives de Médecine Sociale*, Paris).

Prof. Gunnar Dahlberg (Swedish State Institute for Human Genetics and Race Biology).

Sir Raphael Cilento (Director of Health and Medical Services for Queensland, and Professor of Social Medicine, Brisbane).

Dr. Karl Evang (Director General of Public Health, Norway).

Prof. Elena Fambri (Institute of Social Medicine, Rome).

Prof. M. Kacprzak (Government School of Hygiene, Warsaw).

Prof. J. Leclercq (Institute of Forensic and Social Medicine, University of Lille).

Dr. M. G. Neurdenburg (Joint Editor of the *Tijdschrift voor Sociale Geneeskunde*).

Dr. V. J. Prosek (Institute of Social Medicine, Charles University, Prague).

Prof. J. G. Remijnse (Institute of Social Medicine, University of Utrecht).

Prof. Knud Sand (President of the International Academy of Forensic and Social Medicine ; Institute of Social Medicine, Copenhagen).

Prof. Réné Sand (Institute of Social Medicine, University of Brussels).

Dr. Henry E. Sigerist (Retired).

Dr. A. Stampar (Department of Public Health and Social Medicine, Zagreb University, Yugoslavia).

Prof. Wilson M. Wing (School of Hygiene and Public Health, the Johns Hopkins University, Baltimore).